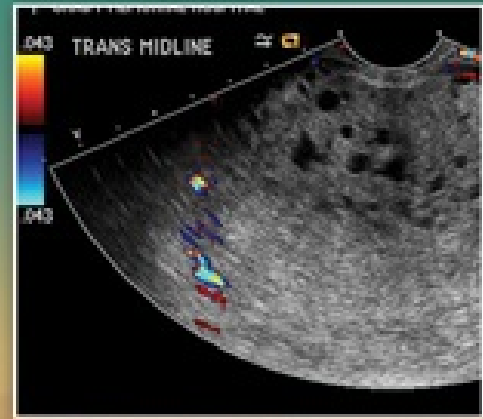


# ATLAS OF Clinical Emergency Medicine



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# Clinical Emergency Medicine

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*To my father, for being an example of courage, compassion, and perseverance.*

—SCOTT C. SHERMAN, MD

*I would like to thank my spouse, Dr. Lane Coffee, for unwavering enthusiasm for and confidence in me during this project and my academic career. I need to thank my parents, Carol (Provci) and Stephen Cico, for their support and encouragement throughout my life and education. I wish to thank my grandparents, Libby (Havlicek) and John Provci, and Margaret (Golobich) and Steve Cico, and especially my great-grandmother “Baba” Anna Nosek Havlicek, who imparted this wisdom on four generations of our family—“People can take everything away from you, but they can never take away your education!”*

—STEPHENJOHN CICO, MD, MEd, FAAEM, FAAP

*To my wife, Jazmir, and children, Rocio, Ambrose, and Cleo, for their enduring love and forbearance of too many fatherless nights and weekends. To my parents for a lifetime of support.*

—ERIK NORDQUIST, MD

*Most importantly, I would like to thank the patients who have allowed photographs of themselves to educate physicians. I also wanted to thank the department for your interest in providing me great photography opportunities. And my family that has stood by me all these years and shown me what is really important in life.*

—CHRISTOPHER ROSS, MD, FRCPC, FACEP, FAAEM

*I would like to gratefully thank my wife Daria, my children—Natalia, Sophia, Julia, and Amelia, and my colleagues for their support in allowing me the time*

*to bring this atlas to life. I also want to thank the medical students and residents that I have had the privilege to teach over the years. You have all made me a better person and physician with your inspiration and collaboration.*

—ERNEST WANG, MD, FACEP



## **FOREWORD**

One of the greatest pleasures in medicine is the art of diagnosing patients' conditions and translating that into an appropriate therapeutic plan. Over the years, the evolution of medicine has increased pressure on practitioners to see more patients in shorter time frames to keep up with the economic demands of modern medicine. This evolution has threatened to turn the art of medicine into the factory of medicine. The *Atlas of Clinical Emergency Medicine* is a tool that practitioners can use to “turn back the clock” during patient encounters. The *Atlas* enhances our ability to instantly recognize patients' conditions through the wide array of images. In addition, the *Atlas* provides us with specific and concise diagnostic and treatment plans. The editorial team consists of outstanding practitioners and educators who care for thousands of patients each year. They have assembled an impressive array of images that enriches our practice of medicine so that we can regain the art of medicine that we yearn for on a daily basis.

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## **PREFACE**

It was 2001 when I began taking photographs in the medical setting. I was in my emergency medicine residency, rotating at a community hospital on the northwest side of Chicago. I bought a disposable “cardboard box” camera on the way into my shift one day. I had no formal training, just an interest in documenting for the purpose of education. I still remember some of the cases from that month—nail gun injury, scapholunate dissociation. I enjoyed it and started giving lectures the following year to my fellow residents with the pictures. It seemed to make it more real to the audience. I graduated to a Canon Elph because it easily fit in the pocket and I dutifully carried it with me on every shift for years to come building a library of images that mostly sat unused on the hard drive of my office computer.

I have always subscribed to the statement that “a picture is worth a thousand words.” It is the way I have best learned throughout my schooling. Seeing a physical finding, radiographic image, or a disease process made it stick in my mind much better than reading pages of voluminous text. And today, in the clinical setting, I prefer to hear a student presentation at the bedside. Not just because it involves the patient and allows for bedside teaching but because I have a visual of the patient and the words take on more meaning. I may not be alone in this belief. Research suggests that approximately 70% of the population uses visual/spatial thinking either predominantly or in combination with words.

In the *Atlas of Clinical Emergency Medicine*, you will find 342 topics laid out in the same, straightforward, organizational format. Immediately under the topic title, the reader will find a relevant

clinical photo, radiograph, or electrocardiogram. For ease of use, text is either below or on the opposing page and is divided into three distinct, clinically relevant sections—“Clinical Presentation,” “Diagnosis,” and “Management.” Our table of contents is driven by the best available clinical photos and guided by the topics listed on the Model of the Clinical Practice of Emergency Medicine. We aimed in each topic to give the clinician just the right amount of information to help them make a diagnosis in the emergency department or study for an exam. In addition to our own libraries of images, the editors have solicited authors from around the country and combed through Wolters Kluwer’s archives to find suitable images for the text.

I could not be more grateful to the patients who I have come across during my career who have allowed their picture to be taken and have granted permission to use it to teach others and ultimately help future patients who have yet to be seen in our clinics and emergency departments.

**Scott C. Sherman, MD**  
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SECTION

A

TRAUMA

SECTION EDITOR  
Ernest Wang

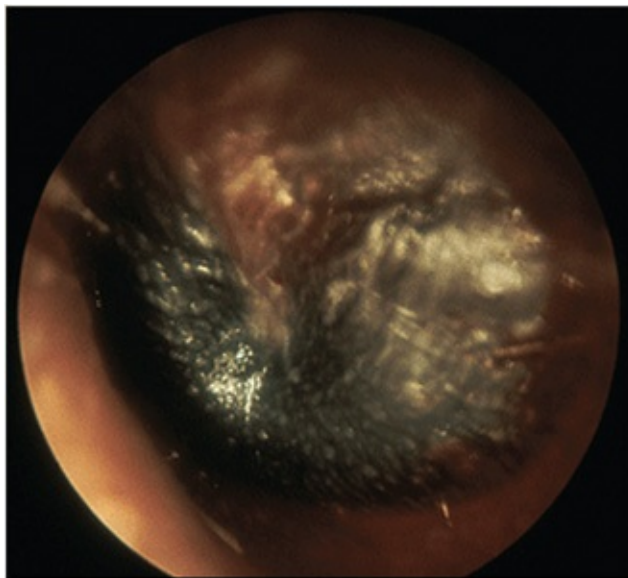


# 1. BASILAR SKULL FRACTURE

Michael Gottlie



**FIGURE 1.** Raccoon eyes. (Reprinted from Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)



**FIGURE 2.** Hemotympanum. (Courtesy of Ellen Deutsch, MD.)

Clinical Presentation

Basilar skull fractures are linear fractures through the base of the skull often involving the temporal bone.

Clinical manifestations may take 6 to 12 hours to fully develop.

Cerebrospinal fluid (CSF) leaks develop in 11% to 45% of basilar skull fractures.

## Diagnosis

Signs of basilar skull fracture include Battle's sign (ecchymosis of the mastoid process), raccoon eyes (periorbital ecchymosis), subconjunctival hematoma, anosmia, cranial nerve deficits, CSF rhinorrhea or otorrhea, and hemotympanum.

CSF rhinorrhea or otorrhea can be identified by testing for glucose and  $\beta$ 2-transferrin levels.

The "halo sign" (placing the blood-tinged fluid onto a piece of tissue paper and assessing for a halo of clear fluid surrounding the blood) has been shown to be unreliable for identifying CSF.

The imaging test of choice is computed tomography with thin cuts through the skull base.

## Management

Most patients are treated conservatively if they remain neurologically intact. Initial treatment involves elevating the head of the bed and supportive care along with early neurosurgical consult.

Despite the potential for meningitis from exposed CSF, prophylactic antibiotics are not routinely recommended.

Indications for surgery include gross contamination, dural tear with pneumocephalus, underlying hematoma, and persistent CSF leak.

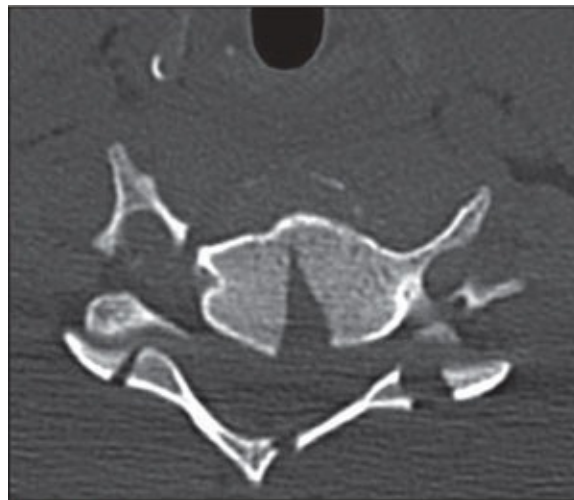
Traumatic carotid cavernous fistula is a rare and potentially deadly complication occurring in 3.8% of cases.

## 2. CERVICAL SPINE FRACTURE

Andrew Agos



**FIGURE 1.** Lateral reconstruction from a CT scan that demonstrates the compression and retropulsion of C6. This high-impact flexion-type injury caused complete disruption of both anterior and posterior columns of C6.



**FIGURE 2.** Axial view of C6 demonstrating the complete disruption anteriorly, posteriorly, and laterally with impingement into the canal.

### Clinical Presentation

Five percent to 10% of unconscious trauma patients will have a cervical spine

injury.

Accounts for 5,000 new cases of quadriplegia and 6,000 deaths in the United States yearly

Fifty percent of injuries at C6 and C7; 35% at C2

Twenty-four percent to 46% incidence of vertebral artery injury, which carries a high morbidity in the elderly if missed

## Diagnosis

Signs include pain, tenderness to palpation, limited range of motion and/or pain on passive motion, and neurologic findings such as weakness, paralysis, or paresthesias.

Cervical spine radiographs are indicated in patients with the above signs or symptoms, patients with altered levels of consciousness either from head trauma or intoxication, and patients with distracting injuries.

Cervical spine series includes an anteroposterior, an open mouth, and a lateral view to the level of C7–T1, assessing disk height and spaces, lateral mass alignment, spinous process alignment, and anterior and posterior lines as well as spinolaminar lines.

Any question on plain films or persistent pain should warrant a computed tomography (CT) scan. In some trauma centers, CT scanning is replacing plain radiographs in high-mechanism traumas and or those with suspected head injury.

## Management

Maintain cervical spine stability with proper immobilization to include a long board, cervical collar, padding, or cushions on both sides of the head for lateral support with tape or straps to secure and stabilize the neck and back.

Improper immobilization during transport can lead to further spinal cord injury in up to 25% of cases.

Identify airway stability and continue to reevaluate during assessment.

Provide in-line stabilization if airway intervention is needed. Do so in a team approach as to maintain neck stability as the collar is loosened.

Be aware of neurogenic shock (hypotension and bradycardia), as persistent

hypotension in the face of spinal cord injury has a worse prognosis. Treat with fluid resuscitation as well as vasopressors.

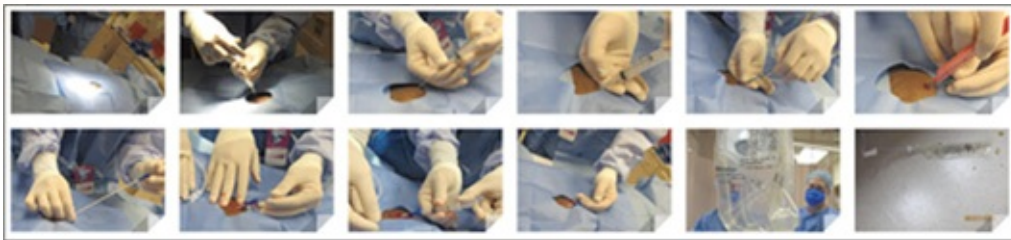
Magnetic resonance imaging is necessary in a hemodynamically stable patient to completely assess and rule out multilevel ligamentous injury.

Surgical intervention for decompression and stabilization



### 3. DIAGNOSTIC PERITONEAL LAVAGE

Paul Bobryshev, Trevor Lewis, Kimberly Nagy



**FIGURE 1.** Closed DPL technique involves inserting a catheter and infusing a liter of saline into the peritoneal cavity and analyzing the fluid that returns after the saline bag is lowered to the ground.

#### Clinical Presentation

A surgical diagnostic procedure to determine if there is free fluid (most often blood) in the abdominal cavity after trauma

The most sensitive test for mesenteric, diaphragm, and hollow viscus injuries

Diagnostic peritoneal lavage (DPL) can be considered in the following hemodynamically stable patients:

Blunt abdominal trauma where computed tomography (CT) or focused assessment with sonography for trauma (FAST) is not available or where imaging is equivocal

Penetrating trauma without obvious indication for operating room (OR)

High suspicion for hollow viscus injury with negative or equivocal imaging

Unreliable abdominal exam (i.e., altered mental status, intubated, spinal cord injury) with negative or equivocal imaging

Changes in abdominal exam or vitals in observed patients with negative initial imaging

Hemodynamically tenuous patients with blunt or penetrating trauma who cannot be safely transported out of the resuscitation bay (i.e., CT scanner, interventions for other injuries)

DPL should not delay emergent laparotomy when it is indicated:

hemodynamically unstable, gunshot wounds, stab wounds with evisceration or peritonitis, or in patients with positive diagnostic imaging (positive FAST or

CT scan identifying an injury requiring surgical intervention).

## Diagnosis

If any of the following are found during aspiration of abdominal contents, then the DPL is considered positive and operative exploration is warranted:

10 mL gross blood or enteric contents

100,000/mm<sup>3</sup> red blood cells

500/mm<sup>3</sup> white blood cells

Presence of bile, bacteria, or food particles

## Management

DPL is performed one of three different ways:

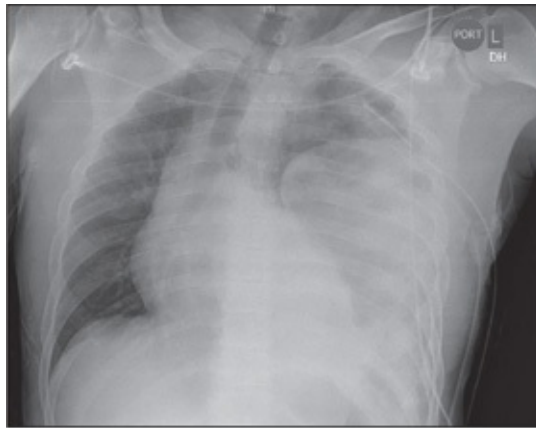
The closed technique is the most common and relies on percutaneous needle access to the peritoneal cavity, followed by the insertion of a catheter using Seldinger technique.

The open technique uses a vertical incision and direct visualization of peritoneal entry with a scalpel. This technique is indicated in patients with pelvic fractures by performing supraumbilical incision and patients with gravid uterus by performing suprafundal incision.

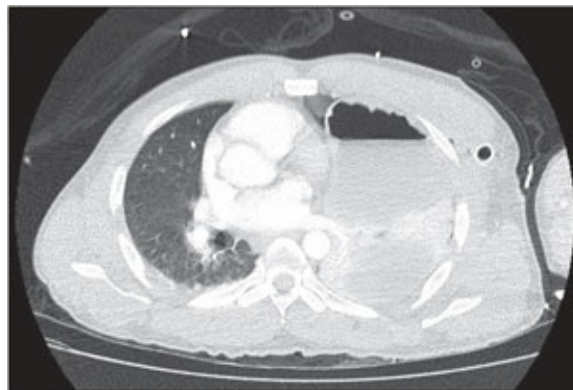
The semiopen technique follows the same principles of the open technique except that the midline fascia is penetrated with a needle and the catheter is advanced using the Seldinger technique. This technique is indicated in obese patients.

## 4. DIAPHRAGMATIC RUPTURE

Gregory Podolej



**FIGURE 1.** CXR demonstrating diaphragmatic rupture.



**FIGURE 2.** Diaphragm rupture (CT).

### Clinical Presentation

Suspect diaphragmatic rupture with penetrating injury to the thoracoabdominal region or other mechanisms with significant, direct-impact forces applied to the abdomen such as falling from height or high-speed motor vehicle accidents.

The left hemidiaphragm is more commonly injured than the right. Not only does the liver provide anatomic protection to the right hemidiaphragm, the right portion of the diaphragm also appears to be more resistant to applied



pressure than the left.

There are no reliable external physical exam findings that portend traumatic diaphragmatic injury from blunt trauma.

Respiratory distress, decreased lung sounds, auscultation of bowel sounds in the thorax, and Kehr's sign (referred visceral shoulder pain) should raise suspicion for diaphragm injury.

## Diagnosis

Chest radiography has low sensitivity and specificity for diaphragmatic injury, as a small rent or hernia is easily masked by a superimposed hemothorax or pulmonary contusion.

Common radiographic findings include herniated abdominal contents such as a displaced mediastinum, herniated intrathoracic bowel loops, gastric bubble, or liver.

Provided the patient is hemodynamically stable, helical computed tomography (CT) scan has both high sensitivity and specificity (94% and 96%, respectively) for the detection of diaphragmatic injury.

Direct visualization of the diaphragm via laparotomy is the gold standard for diagnosis.

## Management

Patients should be resuscitated per Advanced Trauma Life Support protocol.

Given the amount of force required to rupture the diaphragm from blunt trauma, clinicians should have a high suspicion for associated injuries to visceral organs such as liver, spleen, lung, and bowel.

If there are no contraindications, an attempt should be made to place an easily passable naso- or orogastric tube to decompress an intrathoracic bowel hernia to improve lung mechanics and facilitate operative reduction.

All diaphragmatic injuries require operative repair because they are under tension and will not heal spontaneously. A small tear may widen over time, leading to possible long-term complications such as strangulation and incarceration of peritoneal contents. Most diaphragmatic injuries can be accessed and repaired via midline laparotomy.



## 5. EMERGENCY DEPARTMENT THORACOTOMY

James Patrick Hoffman



FIGURE 1. Repair of a cardiac injury.

### Clinical Presentation

Emergency department (ED) thoracotomy is a drastic final effort to save the life of an arresting trauma victim.

The primary goals of the procedure are hemorrhage control, release of cardiac tamponade, facilitation of internal/open cardiac massage, prevention of air embolism, exposure of the descending thoracic aorta for cross-clamping, and repair of cardiac or pulmonary injury.

### Diagnosis

There are two accepted indications for ED thoracotomy:

Penetrating chest trauma with signs of life in the field: traumatic arrest with previously witnessed cardiac activity, unresponsive hypotension (systolic

blood pressure [BP] <70 mm Hg)

Blunt chest trauma with signs of life lost in the ED: unresponsive hypotension (systolic BP <70 mm Hg), rapid exsanguination from chest tube (>1,500 mL)

## Management

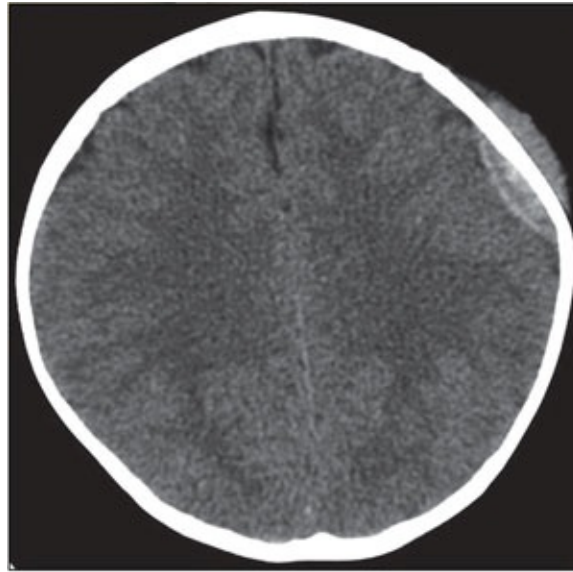
Throughout the thoracotomy procedure, assessment is done to ascertain cause of arrest and interventions performed to correct the cause.

Procedure is always started with left-sided approach for quicker access to the pericardium. Incision is performed from sternum to posterior axillary line in the 4th or 5th intercostal space. After cutting through deep tissues, ribs are spread and lung is pushed away to access the pericardium. Pericardiotomy is performed followed by inspection of myocardium for lacerations. Cardiac defects can be covered with a finger until the wound can be closed with sutures, staples, or insertion of a Foley catheter.

Cardiac massage is performed with one or both hands to provide improved cardiac output. Defibrillation (15 to 30 J) is performed with internal paddles placed on either side of the heart.

## 6. EPIDURAL HEMATOMA

Kristin E. Fontes



**FIGURE 1.** Lens shaped epidural hematoma on the left.

### Clinical Presentation

Epidural hematoma is an uncommon complication from head injury caused by high-pressure arterial bleeding into the potential space between the dura and skull (most commonly a skull base fracture with tearing of the middle meningeal artery).

Classically, there is head trauma with loss of consciousness followed by recovery of consciousness (the “lucid interval”).

Symptoms immediately following the head injury range from brief memory loss to coma with signs of increased intracranial pressure.

Space occupying mass effect of epidural hematoma expansion may lead to midline shift of cerebral structures and transtentorial herniation.

Following the lucid interval, patients may present with various signs and symptoms including headache, vomiting, altered mental status, cranial nerve palsies, hemiparesis, seizures, and Cushing response.

## Diagnosis

Computed tomography of the head is most widely used diagnostic modality and is readily available, but magnetic resonance imaging is more sensitive in detecting an epidural hematoma.

The classic appearance of an acute epidural hematoma is a “lens-shaped” or “convex-shaped” extraaxial enhancing fluid collection that does not cross suture lines.

In the absence of trauma, cerebral angiography may be indicated to evaluate for an underlying vascular lesion.

## Management

Patients with signs of increased intracranial pressure, deteriorating Glasgow coma score (GCS), or coma should be emergently intubated for airway protection.

Definitive treatment for most patients with epidural hematomas is neurosurgical evacuation and hemostasis of the bleeding vessel(s).

When access to a neurosurgeon will be delayed or is unavailable, burr hole evacuation should be considered for patients with deteriorating GCS or coma. Patients taking anticoagulant medications should undergo emergent reversal prior to surgical intervention.

## 7. ESCHAROTOMY

Bilal Khan, Stathis Poulakidas



FIGURE 1. Escharotomy of the lower extremity.



FIGURE 2. Escharotomy of the torso.

### Clinical Presentation

Escharotomy is an emergent, life- and limb-sparing procedure indicated in the setting of full-thickness circumferential and near-circumferential skin burns. Timing varies from several hours to several days after a burn based on clinical signs.



Deep partial-thickness and full-thickness burns destroy elastic layers of skin, and capillary leak causes the underlying tissue to become edematous and swell. Circumferential burns of the extremities and torso, combined with inelasticity caused by the burns, lead to dangerous increases in compartmental pressure.

Circumferential burns on the torso may prevent lung expansion or lead to abdominal compartment syndrome.

## Diagnosis

Circumferential deep partial-thickness or full-thickness burns require frequent monitoring for compartment syndromes or signs of impending vascular compromise.

In the extremities, delayed capillary refill, weak or absent pulses, or decreased pulse oximetry are some indications for escharotomy. However, one should not wait for these signs to perform escharotomies on circumferential extremity burns.

Absence of chest wall expansion, increasing ventilator assistance, or difficulty bag-ventilating patients indicate need for chest wall escharotomy.

## Management

When possible, the escharotomy should be performed by those with experience at the receiving center; however, the procedure should not be delayed once its need is established if the patient cannot be expediently transferred.

Using a scalpel or electrocautery, an incision should be made through the depth of the eschar so as to allow underlying soft tissue to expand. Care should be taken not to cut underlying muscle fascia, tendons, or neurovasculature.

Incisions should be made on the lateral and medial aspects of the extremities. Two incisions may be made on the dorsum of the hand over the 2nd and 4th metacarpal. On the torso, follow the anterior axillary lines from the clavicle to the anterior iliac crests. Horizontal incisions over the costal margin and clavicles should also be made.



To determine adequacy of the procedure, monitor for signs of returned blood flow or chest expansion.

## 8. EVISCERATION

Brent J. Levy, E. Paul DeKoning



**FIGURE 1.** Evisceration following abdominal compartment syndrome due to massive burn injury. (From Lawrence PF, Bell RM, Dayton MT, et al. *Essentials of General Surgery*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

Patients present with penetrating wound to the abdomen, back, pelvis, thorax, or flank.

Can present as isolated injury or associated with multisystem trauma.

Injuries can be relatively subtle, and penetrating wound may be unimpressive.

### Diagnosis

Perform a primary and secondary survey on all trauma patients.

For patients with *anterior* stab wounds, local wound exploration under sterile conditions by a surgical consultant is appropriate. If the anterior fascia is intact, evisceration is not present.

For patients with posterior or flank wounds, additional imaging with

ultrasound or computed tomography is warranted, and serial abdominal and focused assessment with sonography for trauma exams may be useful. Obtain appropriate laboratory studies which include liver function tests, coagulation markers, and type and screen.

## Management

Airway, breathing, and circulation in conjunction with primary and secondary survey.

Establish intravenous access and resuscitate with crystalloid fluids and blood products if necessary.

Surgical consultation and possible exploratory laparotomy.

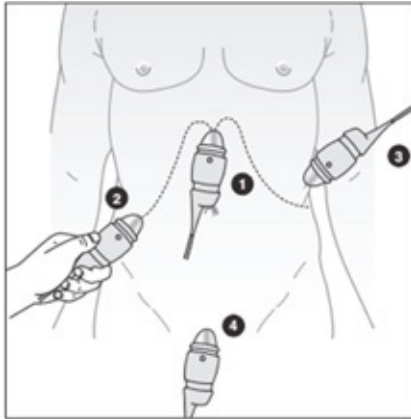
Cover exposed bowel with sterile saline gauze dressing.

Nasogastric tube and urinary catheter placement.

Broad-spectrum antibiotics are indicated with frank evisceration.

## 9. FOCUSED ASSESSMENT WITH SONOGRAPHY IN TRAUMA

Seema Jeelani



**FIGURE 1.** 1 = subxiphoid view; 2 = hepatorenal view; 3 = perisplenic view; 4 = suprapubic view (From Peitzman AB, Rhodes M, Schwab CW, et al. *The Trauma Manual: Trauma and Acute Care Surgery*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

Patients presenting with blunt abdominal trauma and hypotension or patients with stable penetrating abdominal trauma can undergo focused assessment with sonography for trauma (FAST) exams for rapid evaluation of free fluid in the dependent areas of the abdomen.

It is also useful for rapid assessment of cardiac injury in blunt or penetrating chest trauma.

Sensitivity for detecting <400 mL of intraperitoneal fluid is 10% and for 1 L is 97%.

### Diagnosis

Subxiphoid view assesses the pericardial sac by placing the cardiac probe just below the xiphoid process directed cephalad to patients left arm, assessing for black hypoechoic stripe between pericardium and the heart.

Hepatorenal view assesses the hepatorenal recess (Morison's pouch) by placing the abdominal probe between the 11th and 12th rib on the right mid to

posterior axillary line, looking for black hypoechoic stripe between liver capsule and kidney fascia.

Perisplenic view assesses splenorenal recess by placing the abdominal probe between the 10th and 11th rib at the posterior axillary line and assessing for a black hypoechoic stripe between spleen and kidney fascia.

Suprapubic view assesses free fluid in the lower abdomen and pelvis by placing the abdominal probe midline and just superior to the symphysis pubis, looking for a hypoechoic black stripes around the bladder.

## Management

Positive FAST exam in an unstable blunt abdominal trauma requires fluid resuscitation with crystalloid and blood products with likely surgical management.

Positive FAST exam in a stable blunt abdominal trauma will likely require further imaging with an abdomen computed tomography scan or serial FAST exams or surgical intervention and close monitoring for hemodynamic instability.

Positive FAST exam in a stable penetrating abdominal trauma will likely need surgical intervention.

Positive subxiphoid view for pericardial fluid will need a pericardiocentesis if in tamponade or hemodynamically unstable or surgical intervention for pericardial window.

## 10. FULL-THICKNESS BURN

Bilal Khan, Stathis Poulakidas

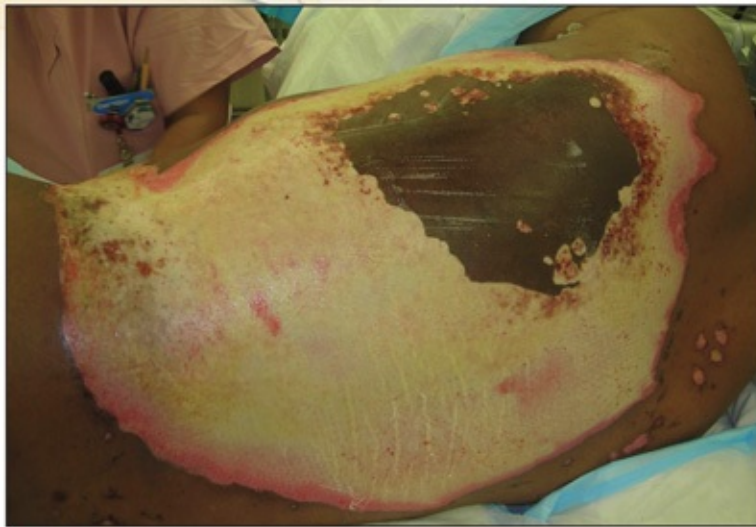


FIGURE 1. Full-thickness burns to the back.

### Clinical Presentation

Full-thickness burns may result from chemicals, electricity, flames, or prolonged contact with hot objects. They may also result from scalding in children and the elderly.

These represent a small minority of burn injuries, although they require more intense intervention.

Concomitant injuries may be common depending on the burn mechanism.

### Diagnosis

Full-thickness burns involve the entire epidermis and dermis and may involve subcutaneous tissue. Muscle, tendon, and bone are spared; burns involving these structures are termed *fourth degree*.

These burns are painless and lack sensation due to nerve damage. They appear white, tan, or brown and are inelastic and leathery. Unlike deep partial-thickness burns, hair follicles will not be spared.

Size should be estimated using the “rule of nines” or an age-specific Lund-Browder diagram. The palmar surface of the patient’s hand (including fingers) represents 1% total body surface area (TBSA). Rule of nines for adults: 9% for each arm, 18% for each leg, 9% for head, 18% for front torso, 18% for back torso; rule of nines for children: 9% for each arm, 14% for each leg, 18% for head, 18% for front torso, 18% for back torso

## Management

Management of patients’ airway, breathing, and circulation must take place prior to assessment of any other injuries. The airway may be compromised regardless of the mechanism of injury secondary to systemic burn edema.

Patients should be assessed for concomitant traumatic injuries.

These patients should also be fluid resuscitated using guidelines such as the Parkland formula.

$4 \text{ mL} \times \text{weight (kg)} \times \text{TBSA (\% burned)} = \text{total mL in 24 hours}$

The first 1/2 is given in the first 8 hours (from the time of the burn).

The last 1/2 is given over the next 16 hours.

All third-degree burns require transfer or urgent referral to a burn center because they will rarely heal without surgical intervention.

As with partial-thickness burns, these wounds should not be debrided or dressed prior to transfer but simply covered with dry, warm sheets. They should be decontaminated in cases of chemical exposure.

Definitive management of nearly all full-thickness burns requires excision and grafting.



## 11. HEMOTHORAX

Jamila Goldsmith



FIGURE 1. CXR with right sided hemothorax.



FIGURE 2. Tube thoracostomy for a hemothorax.

### Clinical Presentation

Most occur secondary to blunt or penetrating chest trauma. Patient may present with hemodynamic instability, respiratory distress, hypoxia, tachypnea, absent or decreased breath sounds, tracheal deviation, or dullness to chest percussion on affected side.



## Diagnosis

Upright chest x-ray (CXR) is diagnostic, although supine film is also acceptable.

Size of hemothorax may affect appearance on CXR; <350 mL appear as small effusion, moderate (350 to 1,500 mL) will appear as opacification of hemithorax, large (>1,500 mL) will have ground glass appearance on supine films.

Computed tomography scan is more sensitive; presence of arterial blush is indication of ongoing bleed and is indication for urgent intervention.

Ultrasound can be performed in patients too unstable for transport to CT scan and will reveal anechoic black region between pleural line and other structures, “wedge” above the diaphragm.

## Management

Once diagnosis is confirmed, tube thoracostomy should be performed by placing a large diameter chest tube (32 to 36 F) in the 6th or 7th intercostal space, midaxillary line, directed posteriorly.

If suspected in an unstable patient, tube thoracostomy should be performed immediately prior to obtaining diagnostic studies.

Surgical intervention should be considered when initial blood loss of 1 to 1.5 L (>20 mL/kg), persistent loss of >200 mL/h (over 2 to 4 hours) or blood transfusion is required.

Autotransfusion is considered for massive hemothorax (>1 L blood loss).

## 12. IMPALEMENT INJURY

Clare Desmond



FIGURE 1. Knife wound to the chest.

### Clinical Presentation

Impalement injury is a broad term that encompasses any object that has penetrated the body.

Injury can range from minor to serious depending on the location and size of the object.

### Diagnosis

Document all pulses and neurologic exam distal and proximal to the injury. X-rays should be performed for extremity injuries; computed tomography (CT) imaging should be performed for all penetrating injuries of the head and

torso, especially if the object is no longer embedded or missing. Do not remove the impaled object, as the embedded object may cause hemostasis or could cause more damage. Large objects and objects impaled in the torso should be removed in the operating room.

## Management

Remove all leveraged parts of impaled objects before imaging. Do not use a handsaw, as the movement can cause further injury.

Consult surgery early.

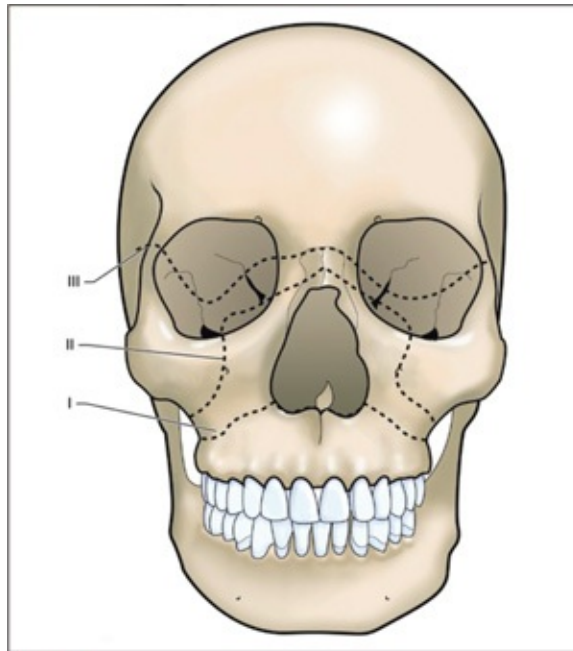
CT angiogram should be performed in extremities with suspicion of vascular injury.

Surgical intervention will be needed in most impalements of the torso and head.

Update tetanus vaccination as indicated.

## 13. LeFORT FRACTURES

Kshiti J. Buch



**FIGURE 1.** LeFort fractures. (From Mancuso AA, Hanafee WN. *Head and Neck Radiology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Fracture of the midface that involve the maxillary dentoalveolar segment and pterygoid plates

Usually seen in association with high-impact, blunt traumatic injuries such as motor vehicle accidents

Usually seen with massive soft tissue swelling, periorbital ecchymosis, bilateral epistaxis, malocclusion, facial lengthening, cerebrospinal fluid rhinorrhea, and cervical and cranial injuries

### Diagnosis

LeFort I—horizontal fracture pattern; fracture of the maxilla through the pterygoid plate; results in disjunction of the maxilla

LeFort II—pyramidal fracture pattern; fracture of nasal bone through the orbital floor; face may appear elongated.

LeFort III—transverse fracture pattern; craniofacial dissociation as the facial bones are separated from the cranial base. Face may appear elongated.

Computed tomography scan of the maxillofacial region, cervical spine, and brain indicated to assess injuries. Chest radiograph also recommended as part of trauma workup and to assess ingested foreign body (i.e., teeth).

## Management

Secure airway as required. Orotracheal intubation is still the preferred method.

Antibiotic coverage if open facial fracture suspected.

Facial trauma consult for definitive open reduction and internal fixation.

## 14. LIVER LACERATION (LIVER FRACTURE/HEMATOMA)

Mark P. Kling

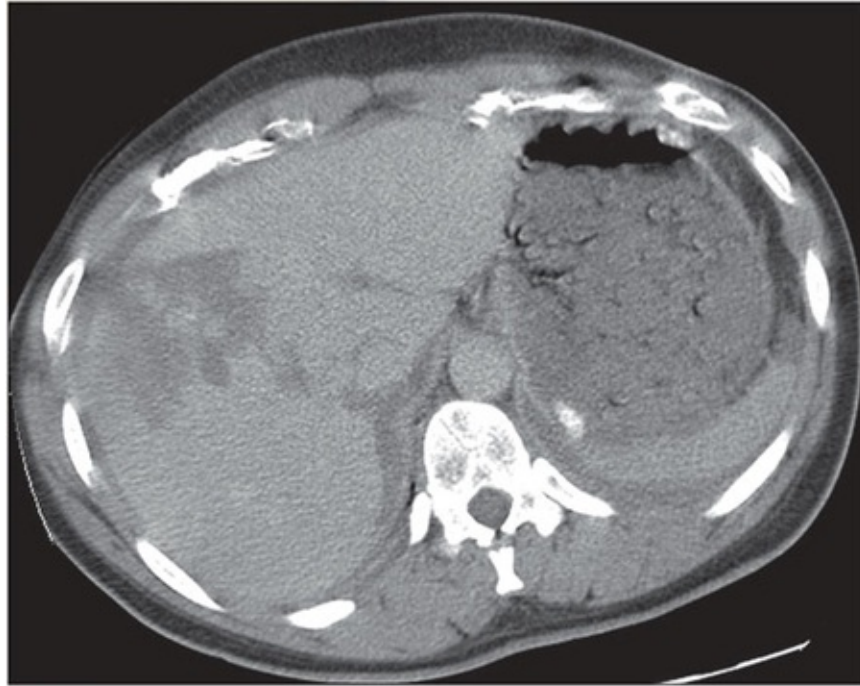


FIGURE 1. Liver laceration (liver fracture/hematoma).

### Clinical Presentation

Most frequently injured abdominal organ

Most common in blunt trauma and second most common in penetrating trauma

Abdominal tenderness specific to right upper quadrant, right rib cage, right flank, or right shoulder from diaphragm irritation

Other findings include wall hematoma, “seat belt” sign, chest injury, splenic injury, pelvic fractures, and spinal cord injury.

### Diagnosis

Computed tomography scan of abdomen and pelvis with oral and intravenous (IV) contrast if possible.

Focused assessment with sonography for trauma (FAST) exam common if hemodynamically unstable; subcapsular, intraperitoneal, or Morrison's pouch fluid indicative

Diagnostic peritoneal aspiration and diagnostic peritoneal lavage can be performed if FAST is equivocal.

Magnetic resonance imaging and magnetic resonance angiography are of limited value and should be performed only if the patient is stable or has an IV contrast allergy.

## Management

Hemodynamically unstable patients require emergent exploratory laparotomy. Hemodynamically stable patients with no other exploration indications should be observed.

Consider use of arteriography and/or embolization.

## 15. LUMBAR SPINE FRACTURE

Andrew Agos



**FIGURE 1.** Less than 50% compression of the L1 vertebral body height.





**FIGURE 2.** Greater than 50% compression of L2 vertebral body height.

## Clinical Presentation

Most common causes are motor vehicle collisions and falls.

Fifty percent of injuries occur in the thoracolumbar spine (T12–L2). The thoracolumbar junction represents the transition point from the rigid lower thoracic spine to a more flexible lumbar spine.

Compression fractures involving the superior endplate are most common.

## Diagnosis

Lower back pain, tenderness, and/or crepitus to palpation of the lumbar spine

Step off or indentation of the spinous process

Neurologic findings to include weakness, paralysis, and paresthesia of the lower extremities from nerve root avulsion or cauda equina syndrome seen with burst fractures with retropulsion.

Lateral radiograph is usually diagnostic. The most common finding is loss of anterior body height. Degenerative changes may limit the visibility of the fracture. If clinically suspected and equivocal radiographs, obtain a CT scan.

If CT imaging confirms a fracture, imaging of the entire spine with CT scanning to rule out multilevel synchronous fractures should be performed.

Up to 20% of patients can have multilevel fractures in the appropriate clinical/trauma setting.

In addition, a magnetic resonance imaging (MRI) of the lumbar spine to evaluate stability.

## Management

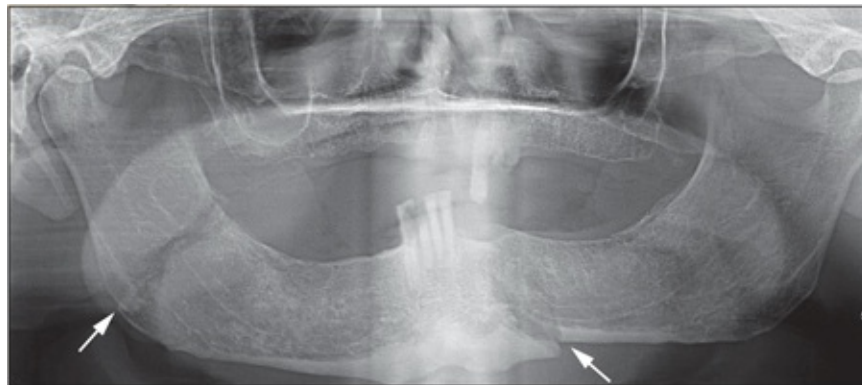
If the MRI does not reveal any lumbar instability or canal impingement, the injury can be managed with a lumbar orthotic provided that there is no neurologic deficit or greater than 50% vertebral body height loss. If there is ligamentous instability or canal compromise, then surgical stabilization and or decompression would be necessary.

## 16. MANDIBULAR FRACTURE

Avani Desai



**FIGURE 1.** Clinical photo of displaced mandibular fracture.



**FIGURE 2.** Mandibular fracture (Panorex).

### Clinical Presentation

Etiology most commonly associated with trauma (assault, motor vehicle accidents, and falls).

Symptoms include mandibular pain worse with jaw movement, decreased bite strength, malocclusion, paresthesia of ipsilateral lower lip in distribution of mandibular nerve branches, periauricular or chin swelling, or ecchymosis.

Evaluation for malocclusion should be performed by instructing the patient to close the mouth and asking the patient to assess for misalignment.

Decreased bite strength can be elicited by having the patient bite down on a tongue depressor while the examiner applies traction to pull it out of the mouth. Inability of the patient to maintain hold on the tongue depressor should raise suspicion of a mandibular fracture.

Physical exam should include careful intraoral examination for mucosal injury or crepitus as well as a palpable step-off or visible separation of lower teeth.

## Diagnosis

If clinical suspicion is low, the initial imaging choice is panoramic radiography. If unavailable, obtain lateral, posteroanterior, and Towne's (condyle) views.

Maxillofacial computed tomography (CT) should be performed if there is a high clinical suspicion of injury or concern for condyle fractures or multiple facial fractures.

Fractures can be unilateral or bilateral with one injury at impact site and a second on the opposite side.

## Management

Assess for airway status.

If stabilization or additional pain control is needed, a Barton's bandage (or ace wrap around head and lower jaw in figure-of-eight pattern) can be applied.

Oromaxillofacial or plastic surgery should be consulted.

If there is an open or unstable fracture, the patient should be admitted for fixation or wiring.

If the fracture is closed and pain is controlled, patient can be discharged to home with soft diet and close outpatient follow-up.

## 17. ORBITAL BLOWOUT FRACTURE

Michael Gottlieb



FIGURE 1. Right inferior rectus muscle entrapment.

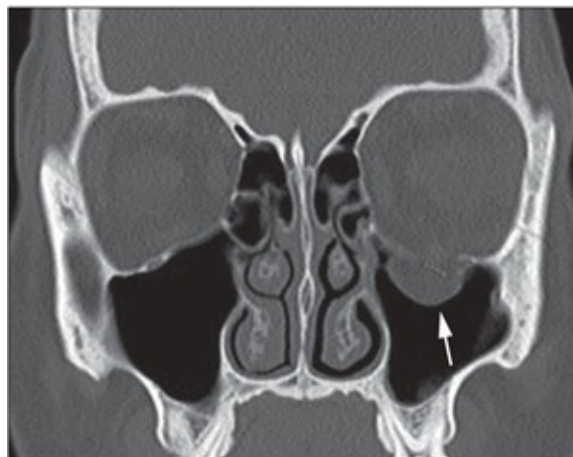


FIGURE 2. Coronal CT demonstrating orbital floor blowout fracture (arrow).

### Clinical Presentation

An orbital blowout fracture involves fracture of the thin medial wall or floor of the orbit.

This is the second most common midfacial fracture, second only to nasal fractures.

The most common etiology is a direct blow to the orbit from blunt head

trauma, often due to a physical altercation.

There is up to a 30% risk of concomitant globe rupture with this injury.

## Diagnosis

Signs of this injury include subconjunctival hemorrhage, periorbital ecchymoses and edema, enophthalmos, diplopia, orbital emphysema, and loss of sensation over the ipsilateral maxilla.

Diplopia on upward gaze suggests entrapment of the inferior rectus muscle.

On a Waters' view radiograph, a polypoid mass can be observed below the orbital floor, classically referred to as the *teardrop sign*.

Computed tomography with thin cuts through the orbit has supplanted radiographs as the test of choice.

## Management

Most blowout fractures heal spontaneously without significant consequence. Prophylactic antibiotics are usually prescribed. Corticosteroids may be administered to reduce swelling.

Patients should be prescribed nasal decongestants and advised not to blow their noses to avoid creating or worsening orbital emphysema.

All patients should follow up with an ophthalmologist.

Surgery is indicated for enophthalmos greater than 2 mm, fracture involving greater than 50% of the orbital floor, and entrapment of extraocular muscles.

However, it is often delayed for more than 2 weeks to let the swelling subside.



## 18. PARTIAL-THICKNESS BURN

Bilal Khan, Stathis Poulakidas



**FIGURE 1.** Partial-thickness burn of the fingers.



**FIGURE 2.** Superficial partial-thickness burn of the chest and upper abdomen.

### Clinical Presentation

Burns involving large total body surface areas (TBSAs) and inhalation injury often lead to significant morbidity and mortality, although the majority of burns are small and superficial and do not require hospitalization.

Most burns are a consequence of accidental mechanisms; however, injuries

resulting from abuse and criminal intent are not uncommon. Partial-thickness burns commonly result from scalding and fleeting contact with hot objects and flames.

## Diagnosis

Assessing the depth of burns on initial presentation is difficult even for the experienced clinician because many wounds progress with time.

Superficial partial-thickness burns involve the entire epidermis and part of the dermis. They are painful, sensitive, appear erythematous and blistered, and blanch with pressure. Deep partial-thickness burns involve deeper layers of the dermis, appear inelastic, and do not blanch.

Size should be estimated using the “rule of nines” or an age-specific Lund-Browder diagram.

## Management

Partial-thickness burns greater than 10% TBSA or burns involving the hands, face, genitals, or joints should be transferred to a burn center.

Greater than 20% TBSA partial-thickness burns should be fluid resuscitated using the Parkland formula ( $4 \times \%TBSA \text{ burned} \times \text{weight in kilogram}$ ).

When transferring burn patients, wounds should not be debrided or dressed because this may interfere with assessment at the receiving center. Cover with warm, dry sheets. In cases of chemical exposure, wounds should be decontaminated prior to transfer.

Patients with small burns not in cosmetically or functionally important areas may be referred and discharged home. Their wounds should be gently cleansed if dirty and dressed with silver sulfadiazine and gauze. Bacitracin should be used on the face and in sulfa-allergic patients. Blisters may be cut away prior to dressing.

Provide adequate analgesia and tetanus prophylaxis prior to transfer or discharge.

Definitive management for superficial partial-thickness burns involves continued physical and enzymatic debridement and application of silver sulfadiazine, silver-impregnated dressings, or other antimicrobial products.



Definitive management of deep partial-thickness burns may also involve skin grafting.

## 19. RETROBULBAR HEMATOMA

Daniel Wachter



FIGURE 1. Proptosis.



FIGURE 2. Lateral canthotomy.

### Clinical Presentation

Blunt trauma to the orbit leads to retrobulbar hemorrhage and edema. Intraocular pressure (IOP) increases causing proptosis, but only to a point because the medial and lateral canthal tendons limit protrusion.

Central retinal artery ischemia can result unless lateral canthotomy is performed.

## Diagnosis

Proptosis

IOP >40 mm Hg (or marked difference on bilateral orbital compression/palpation)

Decreased visual acuity

Restricted extraocular movements

Afferent pupillary defect

## Management

Treatment is with lateral canthotomy with cantholysis.

Lateral canthotomy is contraindicated if there is evidence of globe rupture.

Inject lateral canthus with 1 to 2 mL lidocaine (1% or 2%) with epinephrine.

Devascularize tissue by placing hemostat on lateral canthus directed toward the lateral bony orbit for 30 to 90 seconds.

Remove hemostat and cut devascularized tissue 1 to 2 cm with scissor.

Expose then sever the inferior lateral canthal tendon.

Expose then sever superior lateral canthal tendon in same fashion if IOP remains >40 mm Hg after inferior cantholysis.

Emergent ophthalmology referral to assess for retinal ischemia and to manage recovery

## 20. SEAT BELT SIGN

Kelly Bailey, Colleen Crowe

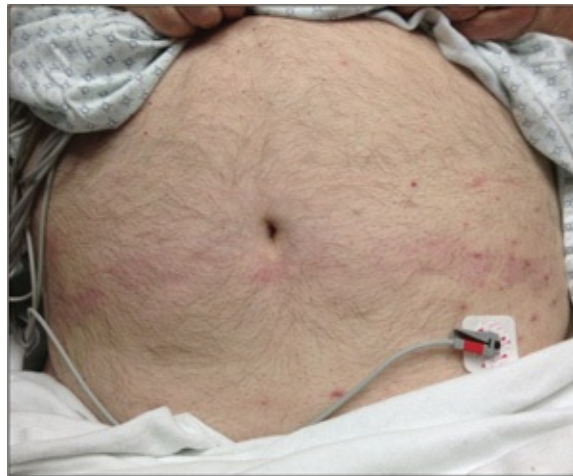


FIGURE 1. Ecchymosis across the abdomen.



FIGURE 2. Ecchymosis across the shoulder.

### Clinical Presentation

An abrasion, contusion, or ecchymosis over the abdomen, chest, and/or neck that is present along the distribution of the seat belt

Occurs from contact of the body against the seat belt during a motor vehicle

collision

Associated with various injuries including vascular (i.e., carotid, vertebral, aortic, iliac, innominate, or subclavian), fractures (ribs, clavicular, sternal, chance), tracheal or laryngeal injury, pulmonary contusion, abdominal wall disruption, hollow viscous injury, mesenteric injuries, solid organ trauma. Many injuries are associated with improper use and positioning of the seat belt.

## Diagnosis

Obtain computed tomography angiography to assess for a vascular injury if any of the following are present: C1–C3 fracture, cervical spine fracture with subluxation, fractures involving foramen transversarium, or clinical findings suggestive of injury to the neck (abrasions/ecchymosis).

Obtain anteroposterior chest x-ray for initial evaluation of the thorax. Obtain a chest computed tomography (CT) for further evaluation if there is radiologic or clinical evidence suggestive of thoracic trauma.

Perform a FAST exam to assess for free intraperitoneal fluid. This exam may be repeated in 30 minutes in patients with no initial findings.

Order an abdomen/pelvis CT with contrast in a hemodynamically normal patient who does not have obvious indication for emergent laparotomy.

Obtain cervical spine CT if midline neck pain, midline tenderness on palpation, neurologic deficits associated with cervical spine injury, altered level of consciousness, significant mechanism with distracting injury, or intoxication.

## Management

A patient with seat belt sign may require hospitalization for further observation despite negative initial workup because early ultrasound and CT imaging may not detect subtle injuries.

Patients with blunt carotid or vertebral artery injury require anticoagulation or antiplatelet therapy in consultation with vascular surgery.

Patients with potential intra-abdominal injuries necessitate consultation by a surgeon.

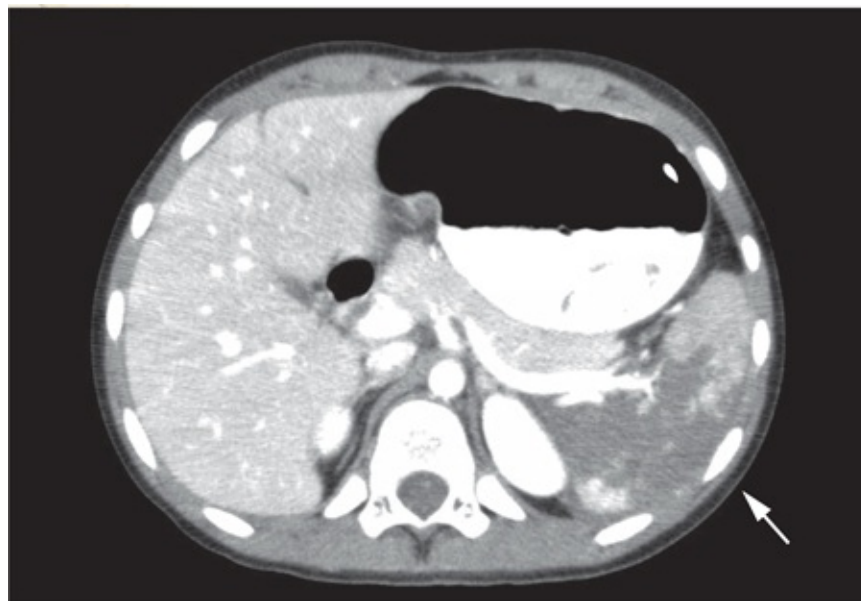


## 21. SPLENIC RUPTURE

Gregory Podolej



**FIGURE 1.** Coronal CT image of splenic rupture (arrow).



**FIGURE 2.** Axial CT image of splenic rupture (arrow).



## Clinical Presentation

Suspect splenic injury or rupture based on mechanism, that is, blunt abdominal trauma from a motor vehicle collision, assault with trauma to the left upper quadrant, or penetrating injuries to the left thoracoabdominal area. About one-third of blunt abdominal trauma patients presenting to the emergency department will have a splenic injury.

The spleen is the most common traumatically injured intra-abdominal organ in children. Initially, children can compensate for acute blood loss and thus can mask the extent of their injuries.

## Diagnosis

The clinician should assess for external signs of abdominal trauma such as localized bruising, the “seat belt sign,” tenderness over the left lower ribs, or penetrating injury to the left upper quadrant.

The focused assessment with sonography for trauma (FAST) examination with special attention paid to the splenorenal fossa should be performed in hemodynamically unstable patients. A negative FAST does not exclude injury or the need for laparoscopy.

Aspiration of gross blood (at least 10 mL) or >10,000 red blood cells per high-power field via diagnostic peritoneal lavage (DPL) indicate the need for laparotomy.

In stable patients, abdominal computed tomography (CT) scan with intravenous contrast is both highly sensitive and specific for splenic injury.

## Management

As with all blunt abdominal traumas, patients should be resuscitated per Advanced Trauma Life Support guidelines. Careful attention should be paid to volume resuscitation with crystalloid and blood products as needed.

Hemodynamically unstable patients with hemoperitoneum diagnosed via FAST or DPL warrant laparotomy and possible splenectomy.

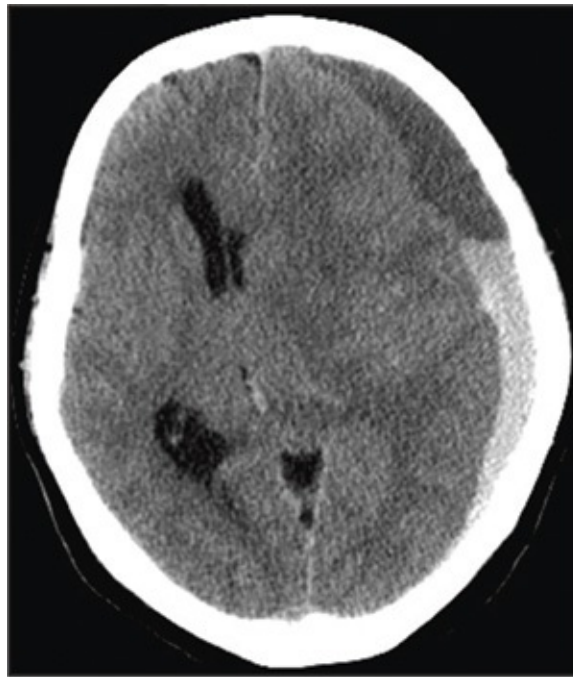
Stable patients with splenic injury as evidenced by contrast blush, splenic hematoma, or laceration on CT have increasingly been managed



nonoperatively, that is, via arterial embolization. Ultimately, further patient management decisions depend on factors such as the extent of organ injury and available resources. They should be made in conjunction with both surgical and interventional radiology consultants.

## 22. SUBDURAL HEMATOMA

Brent J. Levy, E. Paul DeKoning



**FIGURE 1.** CT scan of acute (white) and chronic (black) blood in a patient with left subdural hemorrhage. Note the obliteration of the ventricles and midline shift.

### Clinical Presentation

Caused by acceleration/deceleration of the brain resulting in disruption of bridging veins and can present as acute (<14 days) or chronic (>14 days) following traumatic event

Hematoma develops between the dura mater and arachnoid space, and symptoms result from compression of other structures within the skull; it can result in increased intracranial pressure (ICP) and herniation.

More common in patients with atrophied brain matter (alcoholics, elderly, etc.)

Symptoms include headache, confusion, vertigo, nausea, loss of consciousness, hemiparesis, altered mental status, gait instability, or seizures.

Significant association with use of anticoagulant and antiplatelet agents

## Diagnosis

Perform a complete history and physical exam in order to exclude alternative causes (i.e., toxicology, medication problems, metabolic abnormalities, etc.). Computed tomography (CT) without contrast is the initial imaging study of choice for acute bleeding. Magnetic resonance imaging is more sensitive, better characterizes structures in the posterior fossa, and is indicated in the evaluation of chronic and subacute subdurals.

Initial evaluation should include prothrombin time, partial thromboplastin time, international normalized ratio, complete blood count, chemistry panel, glucose level, and therapeutic drug levels.

## Management

Maintain airway, which may include need for intubation.

Elevate the head of bed to facilitate venous outflow and consider pharmacologic agents to decrease cerebral edema and ICP. This may include hypertonic saline or mannitol.

Neurosurgical consultation for possible surgical decompression is warranted in all cases.

Perform thorough secondary and tertiary trauma survey to rule out additional injuries.



SECTION

B

NEUROLOGIC

SECTION EDITOR

Scott C. Sherman

# 1. ABDUSCENS NERVE PALSY

Ezaldeen Ahmad Numur, Patrick G. Meloy



FIGURE 1. Abducens nerve palsy. (Images courtesy of Jason Peragallo, MD.)

## Clinical Presentation

Paralysis of the lateral rectus muscle causes unopposed adduction of the ipsilateral eye resulting in a convergent strabismus, or esotropia, resulting in diplopia.

Diplopia is made worse with lateral gaze, and patients may adopt a face turn toward the affected eye to compensate.

Unilateral abducens nerve palsy is the most common of the isolated ocular motor nerve palsies found often in older patients with diabetes and/or hypertension.

Inquire about other neurologic signs and symptoms (e.g., headache), which would suggest a secondary cause that would require a more extensive workup.

The abducens nerve is the first cranial nerve compressed with elevated intracranial pressure (ICP); therefore, papilledema, headaches, or meningismus in association with a sixth nerve palsy suggest an elevated ICP.

## Diagnosis

In patients older than age 50 with diabetes and/or hypertension and an isolated sixth nerve palsy, microvascular infarction of the nerve is the most common

cause. The differential diagnosis of secondary causes includes trauma, aneurysm, vascular disease, increased ICP, vasculitis, cavernous sinus mass, neoplasm, multiple sclerosis, meningitis, and thyroid eye disease. Thorough neurologic examination is important to check for additional findings that would suggest a secondary cause.

## Management

In patients older than 50 with diabetes and/or hypertension who have an isolated abducens nerve palsy, imaging in the emergency department is not required. Prompt follow-up should be obtained.

Younger patients (<50 years old) or patients with additional neurologic findings require neuroimaging, neurology consultation, and possible admission searching for secondary causes.



## 2. BOTULISM

Ezaldeen Ahmad Numur, Patrick G. Meloy



**FIGURE 1.** Infantile botulism. (Image provided courtesy of the Centers for Disease Control and Prevention.)

### Clinical Presentation

*Clostridium botulinum* is an anaerobic, spore-forming bacterium that causes a toxin-mediated, rare, but life-threatening illness characterized by flaccid paralysis.

The neurotoxin inhibits acetylcholine release at the neuromuscular junction so there is neither a sensory deficit nor pain associated with the weakness.

Foodborne botulism is the most common form and often results from improperly preserved canned foods. Onset of symptoms is 6 to 48 hours after ingestion of tainted food.

Classic presentation: descending, symmetric paralysis with involvement of the cranial nerves and bulbar muscles first—diplopia, dysarthria, and dysphagia

Infantile botulism occurs from ingestion of spores that are able to produce toxin in the gastrointestinal tract. Presents as constipation, poor feeding, lethargy, and weak cry resulting in a “floppy baby.”

## Diagnosis

Diagnosis based on clinical exam findings (e.g., symmetric descending flaccid paralysis, starting with cranial nerves) and exclusion of other disease processes

Blood and stool testing is performed, and *C. botulinum* culture is used for evaluation of suspected food.

Lumbar puncture should be considered to rule out other disease processes.

## Management

Treatment is generally supportive, and any patient with clinical suspicion of botulism requires admission to an intensive care unit for monitoring.

Consider early intubation for patients because rapid progression to respiratory failure is common.

Horse-serum antitoxin should be administered in adults.

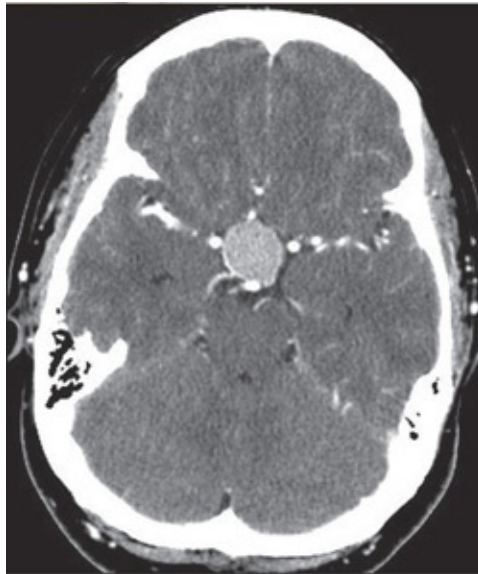
Human botulism immunoglobulin, or “BabyBIG,” can be administered to infants.

Antibiotics should be held because they are likely to cause cell lysis and increase release of the toxin.



### 3. BRAIN TUMOR

Brent Allen, Patrick G. Meloy



**FIGURE 1.** Pituitary tumor on CT.

#### Clinical Presentation

Initial presentation may consist of vague symptoms and include headaches, nausea, vomiting, generalized or focal weakness, new-onset seizures, and cranial nerve defects.

“Morning headaches,” new-onset headaches in middle-aged adults, headaches worsening with Valsalva maneuvers, or headaches with associated visual and olfactory changes should raise suspicion for neoplasm.

Due to the enclosed space of the brain, tumor growth can cause edema, hydrocephalus, and increased intracranial pressure (ICP), all of which can manifest as papilledema, focal and generalized deficits, and altered mental status.

Symptoms are similar in both primary and metastatic lesions, although primary tumors are more common in pediatrics and geriatrics, and metastatic tumors are more common in adults.

## Diagnosis

As described, nonspecific symptoms abound and providers should maintain a high index of clinical suspicion. Specific physical findings depend on the location of the mass and include hemianopsia, anosmia, diplopia, or papilledema.

Noncontrast head computed tomography (CT) should be the first imaging modality in the emergency department (ED), and it may show a space-occupying lesion or areas of hypodensity. Head CT with contrast may demonstrate contrast-enhanced rings. Magnetic resonance imaging has the highest specificity in identifying lesions, although this is not usually obtained in the ED.

Avoid lumbar puncture in patients with suspected neoplastic lesions because this can lead to herniation secondary to increased ICP.

## Management

Intubation may be required in patients who are obtunded and unable to protect their airway. Consider adjunct medications to blunt the rise in ICP. After intubation, if acute herniation is suspected, a brief interval of hyperventilation may be required until bedside neurosurgical involvement.

In patients with elevated ICP, steroid treatment with dexamethasone is recommended.

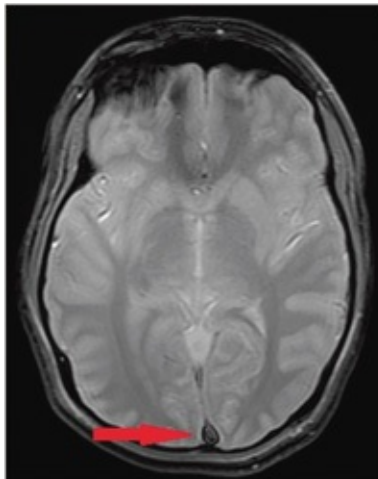
Most patients will require admission if the neoplasm is diagnosed primarily in the ED, and specific treatment options will be formed by the neurosurgery, oncology, and radiation oncology services.

## 4. CAVERNOUS VENOUS THROMBOSIS

Brent Allen, Michelle D. Lall



**FIGURE 1.** Dense signal in the superior sagittal sinus, called the "delta sign" (arrow). (Image courtesy of Tarek Hanna, MD.)



**FIGURE 2.** Lack of sinus filling on MRI (arrow). (Image courtesy of Tarek Hanna, MD.)

### Clinical Presentation

Presence of thrombosis in the dural venous sinuses, which drain blood from the brain

Patients may present with nonspecific symptoms, including recurrent

headache, nausea, vomiting, new-onset seizures, hemiparesis, and cranial nerve defects.

Seen as a complication of closed head trauma, nasal or facial trauma, infectious process, postlumbar puncture, and idiopathic intracranial hypertension

Hypercoagulable states associated with protein C and S deficiencies, pregnancy, malignancy, lupus anticoagulant, and factor V Leiden increase the risk of cavernous sinus thrombosis.

## Diagnosis

In the appropriate context, noncontrast head computed tomography (CT) should be the first imaging modality and may show a “delta sign” or dense signal in the superior sagittal sinus ([Fig. 1](#)).

Head CT with contrast may conversely show the “empty delta sign,” corresponding to the enhancement of collateral veins to the sagittal sinus and lack of enhancement within the sinus itself.

Magnetic resonance imaging (MRI) may show lack of sinus filling ([Fig. 2](#)).

Magnetic resonance venogram is the modern “gold standard” and can directly visualize the thrombus itself while potentially showing dilated venous collaterals or small areas of hemorrhage.

## Management

Systemic anticoagulation has shown benefit for all venous thrombosis patients, including those with known hemorrhage.

There is no known therapeutic difference between unfractionated heparin and low-molecular-weight heparin.

Catheter-directed thrombolytic therapy has been used with some success.

If there is concern for an infectious process, aggressive therapy should be initiated, including broad-spectrum antibiotics for suspected frontal sinusitis and meningitis.

All patients with confirmed or suspected venous sinus thrombosis should have emergent consultations with neurology, neurosurgery, and the intensive care unit. Infectious disease consultations for those patients with concurrent

infections should be considered.

## 5. CEREBELLAR HEMORRHAGE

Sierra Beck

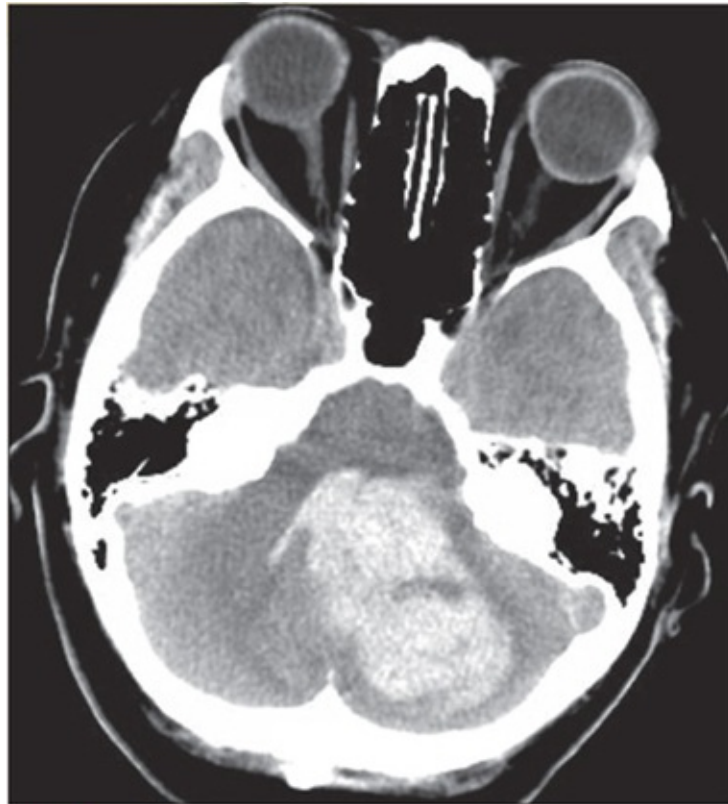


FIGURE 1. Cerebellar hemorrhage.

### Clinical Presentation

Patients with cerebellar hemorrhage present with sudden onset of severe headache, vertigo, vomiting, and ataxia. Clinical course is unpredictable, and some patients may rapidly progress to coma and death if there is associated brain stem compression.

Exam may demonstrate ipsilateral limb ataxia and dysmetria or ipsilateral cranial nerve VI palsy if there is associated brain stem compression.

Risk factors include increasing age and hypertension.

### Diagnosis

Emergent noncontrast computed tomography of the head is the initial diagnostic study of choice.

Cerebellar hemorrhage will appear as hyperdense foci within the cerebellum. Larger hemorrhage (>3 cm), associated brain stem compression, intraventricular hemorrhage, or obstructive hydrocephalus are poor prognostic indicators.

## Management

Patients with low Glasgow coma score require early intubation for airway protection.

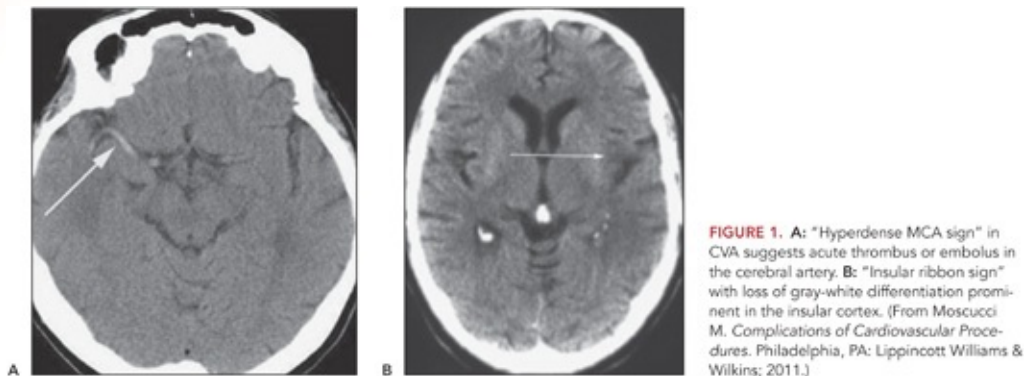
Assess for history of anticoagulant use, check for abnormal coagulation studies, and rapidly reverse any abnormalities.

Early neurosurgical consultation is indicated because patients with cerebellar hemorrhage are more likely than other patients with intracranial hemorrhage to require decompressive craniectomy and hematoma extraction.



## 6. CEREBROVASCULAR ACCIDENT

Cynthia Leung



### Clinical Presentation

Acute onset of neurologic deficit(s) attributable to disruption of blood flow in a particular cerebrovascular distribution

Common clinical syndromes include the following:

Anterior cerebral artery: unilateral weakness and/or sensory loss of contralateral lower extremity greater than upper extremity

Middle cerebral artery (MCA): unilateral weakness and/or sensory loss of contralateral face and upper extremity greater than lower extremity with either aphasia or neglect

Posterior cerebral artery: unilateral visual field deficit in both eyes (homonymous hemianopsia)

Posterior circulation syndromes usually have multiple deficits, which typically include contralateral weakness and/or sensory loss in combination with ipsilateral cranial nerve palsies. Symptoms may include diplopia, dysarthria, dysphagia, facial droop, vertigo, ataxia, nausea, vomiting, and decreased level of consciousness.

### Diagnosis

Diagnosis is based on clinical presentation. The National Institutes of Health



Stroke Scale (NIHSS) provides a standardized clinical assessment, which allows monitoring of the patients neurologic deficits over time.

Workup includes head computed tomography (CT) to exclude intracranial hemorrhage or structural brain lesion, blood glucose, and electrocardiogram.

Most common CT finding in acute ischemic stroke is normal brain. However, multiple subtle findings associated with acute ischemic stroke may be present in the first 3 hours after symptom onset.

## Management

Determine eligibility for thrombolytic therapy. Inclusion criteria include ischemic stroke causing measurable neurologic deficit, <3 hours (4.5 hours in selected patients) from symptom onset, age >18 years. Multiple exclusion criteria must be assessed before thrombolytics are considered.

Recombinant tissue plasminogen activator is currently the only FDA-approved therapy for acute ischemic stroke.

Complete blood count, chemistries, INR, aPTT, and cardiac markers are recommended; however, administration of thrombolytics should not be delayed for results of coagulation studies unless a bleeding disorder is suspected.

Prevent further neurologic deterioration by providing supportive care: airway and ventilatory support, prevention of hyperthermia, cardiac monitoring, blood pressure control, and glucose control.

Goals for blood pressure (BP) control: If getting tissue plasminogen activator (tPA), SBP <180 mm Hg, DBP <105 mm Hg; if not getting tPA, SBP <220 mm Hg, DBP <120 mm Hg. Avoid rapid fluctuations in BP and hypotension.

## 7. EPIDURAL ABSCESS

Michael D. Zwank



**FIGURE 1.** Epidural abscess seen at the T2 level on MRI (arrow).

### Clinical Presentation

Most common presenting symptom is back pain.

Classic triad of fever, back pain, and neurologic deficit is not always present at the time of initial presentation.

Risk factors include elderly, immunocompromise, intravenous (IV) drug use, alcohol abuse, recent instrumentation (dental, gastrointestinal [GI], etc.), or trauma.

Most common bacteria is *Staphylococcus aureus*.

### Diagnosis

Physical exam may demonstrate signs of neurologic compromise including urinary retention and saddle anesthesia.

Magnetic resonance imaging (MRI) is imaging modality of choice.

Inflammatory markers such as white blood cell, erythrocyte sedimentation rate, and C-reactive protein are nonspecific.

Blood cultures can help identify causative organism.

## Management

Early antibiotics are important with broad coverage including methicillin resistant *S. aureus*.

This is a neurosurgical emergency that requires early surgical decompression.

Treat associated sepsis with aggressive IV fluids and supportive care.

## 8. FACIAL NERVE (BELL) PALSY

Tamara Halaweh, Andrew King



**FIGURE 1.** Facial nerve palsy on the right. Note the inability to raise the lips and eyebrows or to fully close the eye.

### Clinical Presentation

Facial nerve palsy is manifested by paralysis of the facial muscles, sometimes with loss of taste on the anterior two-thirds of the tongue.

Typically preceded by a viral illness, with herpes simplex as the most commonly accepted cause.

## Diagnosis

Peripheral nerve involvement is diagnosed when there is weakness of the facial muscles in the upper (eyebrows and forehead) and lower face (mouth).

If the forehead is spared, an intracranial process should be suspected.

Onset is progressive over 1 to 2 days, reaching maximal clinical weakness within 3 weeks.

Ramsay Hunt syndrome is herpes zoster infection of the ear. Patients will present with vesicles in the ear canal.

## Management

Prednisone (60 mg per day) for 7 days is administered in patients who present within 3 to 4 days of onset.

Valacyclovir (1,000 mg three times daily) for 1 week in patients with severe facial palsy at presentation. Acyclovir 400 mg five times daily can also be used, although some studies show that it is no more effective than prednisone alone.

Artificial tears and taping the eye shut at night is recommended.

## 9. HORNER SYNDROME

Mary Wittler



**FIGURE 1.** Ptosis, miosis, and anhidrosis on the left. (From Sherman SC, Bellinger MH. Man with dizziness and vomiting. Horner's syndrome. *Ann Emerg Med.* 2013;62[5]:547, 553.)

### Clinical Presentation

Classic triad is unilateral ptosis, miosis, and facial anhidrosis.

Results from ipsilateral interruption of the sympathetic innervation to the face and eye

Supplied by a three-neuron pathway. The first neuron starts in the hypothalamus and ends in the spinal cord near T1. The second neuron exits the spinal cord, travels near the lung apex, and ends in the superior cervical ganglion (located near the bifurcation of carotid artery). The third neuron travels to facial structures (eyelid retractor [Muller's] muscle, pupillary dilator muscle, and facial sweat glands).

Numerous etiologies exist, including strokes; brain, spinal cord, and lung neoplasm; infection; multiple sclerosis; trauma or surgery to upper chest or neck; carotid or vertebral artery dissections; central venous access; lung apical processes; and tube thoracostomy.

## Diagnosis

The pattern of presentation is dependent on the lesion location and may occur anywhere along the sympathetic pathway from the hypothalamus to the target facial structures; incomplete presentations may occur.

Anisocoria should be worse in the dark compared to in light (owing to lack of sympathetic innervation).

Additionally, a dilation lag is present—the affected pupil dilates slower than the normal pupil in the dark.

Other clinical signs and symptoms suggest lesion location.

## Management

Etiologies include benign and malignant processes, and management varies. Horner syndrome requires thorough clinical evaluation to determine the most likely site of the lesion and to guide diagnostic evaluation.

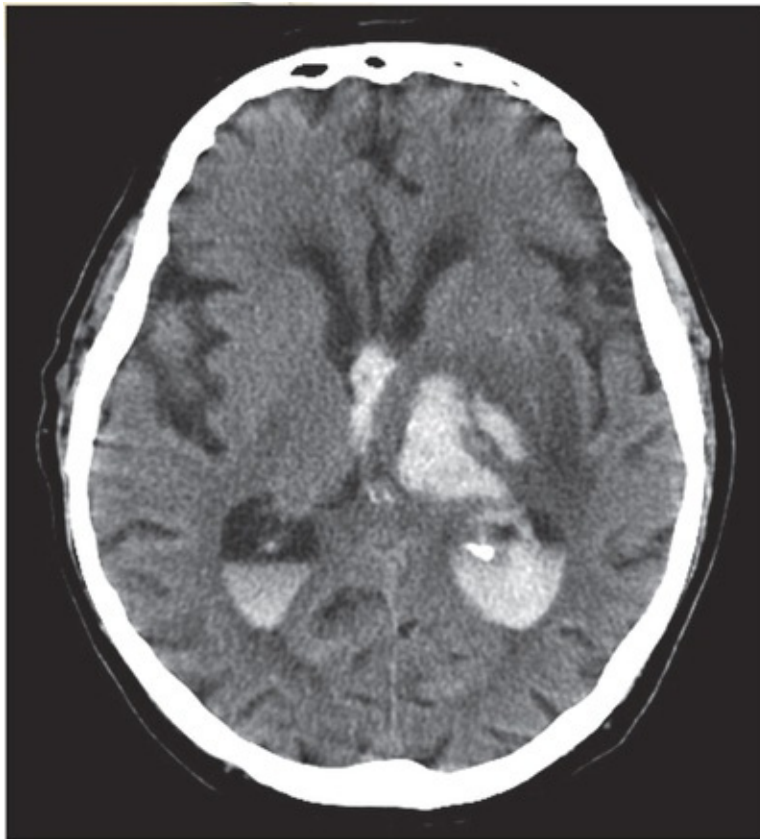
Emergency department evaluation may include a chest x-ray, computed tomography +/- magnetic resonance imaging brain and/or cervical spine, and/or computed tomography angiography +/- magnetic resonance angiography head and neck.

Consultation with neurology is recommended.



## 10. INTRAPARENCHYMAL HEMORRHAGE

David Manthey



**FIGURE 1.** Intraparenchymal hemorrhage CT.

### Clinical Presentation

Most commonly due to hypertensive damage to vessels

Basal ganglia (40% to 50%), temporal lobe (20% to 50%), thalamus (10% to 15%), pons (5% to 12%)

In young patients, vascular malformations are a common cause.

Glioblastomas are most common primary tumor to hemorrhage.

Hemorrhagic transformation occurs in approximately 8% to 13% of all ischemic strokes.



## Diagnosis

No way to distinguish between hemorrhagic versus nonhemorrhagic cause of a focal deficit on history or clinical exam.

Computed tomography (CT) scan without contrast will detect most intraparenchymal bleeding.

## Management

Reversal of any coagulopathy with platelets, factor concentrate, fresh frozen plasma, etc.

Blood pressure control is targeted at 140/90 mm Hg.

Elevate the head of the bed to 30°.

Initiate antiepileptic for seizure activity or lobar hemorrhage. Consider risk/benefit in others.

If impending herniation, hyperventilate and consider mannitol.

Contact neurosurgery for operative options and intracranial pressure monitoring.

## 11. MYASTHENIA GRAVIS

John P. Gaillard



**FIGURE 1.** Simpson test: Ptosis after sustained upward gazing activity.

### Clinical Presentation

Muscle weakness that worsens with repeated use and improves with rest  
Ptosis, jaw fatigue with chewing, dysphagia, nasal speech  
Proximal muscle weakness: arms more often than legs  
Respiratory failure = myasthenia crisis

### Diagnosis

Acetylcholine receptor antibodies  
Muscle-specific receptor tyrosine kinase antibodies  
Tensilon test: Symptoms dissipate after edrophonium (up to 10 mg intravenous). Have atropine at bedside due to potential bradycardia.

### Management

Oral acetylcholinesterase inhibitor: pyridostigmine 30 to 60 mg every 4 to 6 hours

Prednisone 1 mg/kg/d

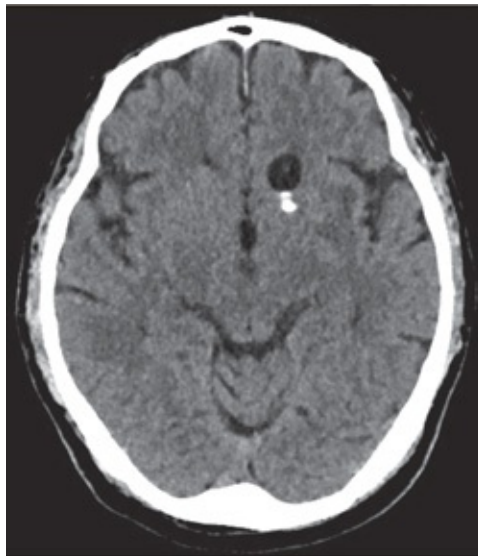
Myasthenic crisis: elective intubation when forced vital capacity <20 mL/kg or negative inspiratory force <-20 cm H<sub>2</sub>O

Plasma exchange, intravenous immunoglobulin

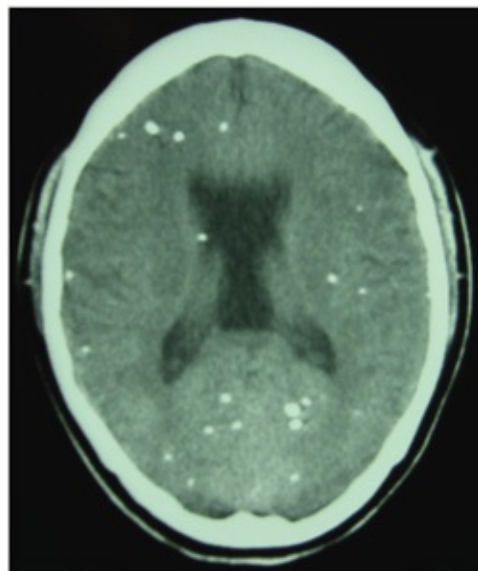
Immunosuppressant therapy (azathioprine, mycophenolate mofetil, cyclosporine) if resistant to steroids

## 12. NEUROCYSTICERCOSIS

Brent Allen, Michelle D. Lall



**FIGURE 1.** Live cyst on brain CT. (Image courtesy of Tarek Hanna, MD.)



**FIGURE 2.** Calcifications from old disease (dead parasites) in a patient with recurrent seizures.

Clinical Presentation

Clinical manifestations depend on the locations of the lesions in the brain, and include seizures, headache, dizziness, stroke, and neuropsychiatric dysfunction.

Caused by ingestion of eggs of *Taenia solium* (pork tapeworm), usually by contaminated food

It is the most common cause of parasitic disease of the nervous system and one of the most common causes of seizures in developing countries.

Endemic in Central and South America, sub-Saharan Africa, and some regions of the Far East

## Diagnosis

Noncontrast head CT will show focal areas of edema. Contrast-enhanced scans will show characteristic “ring-enhancing lesions” or “mural nodules.”

Cerebrospinal fluid analysis from a lumbar puncture may show increased inflammatory markers and lymphocytosis, but this is neither sensitive nor specific for the disease.

Funduscopy examination may reveal intraocular larvae, which are diagnostic.

Lab studies are frequently unhelpful in the emergency department, although serology with enzyme-linked immuno assay for *Taenia* antigens is both sensitive and specific.

## Management

If the parasite is dead (calcified cysts on imaging), the treatment is directed primarily toward symptom management.

If the parasite is active and the patient has signs of vasculitis, arachnoiditis, or encephalitis, the patient should be treated with a course of steroids and anticonvulsants to reduce inflammation surrounding the cysts and lower the risk of seizures.

A first-line antihelminthic, such as albendazole, is indicated, along with symptomatic treatment.

Indications for surgery include hydrocephalus due to an intraventricular cyst, multiple cysts in the subarachnoid space, or obstruction due to arachnoiditis.

Patients with active disease require admission to initiate therapy and may

require consultation with infectious disease, neurology, and neurosurgery.

## 13. OCULOMOTOR NERVE PALSY

Ezaldeen Ahmad Numur, Patrick G. Meloy



FIGURE 1. Oculomotor nerve palsy of the right eye. (Images courtesy of Jason Peragallo, MD.)

### Clinical Presentation

Initial complaint is usually diplopia, either horizontal or vertical.

The eye is deviated down and out secondary to paralysis of the superior rectus, inferior rectus, medial rectus, and inferior oblique.

There is accompanying ptosis and mydriasis, secondary to paralysis of the levator palpebrae, ciliary muscle, and iris sphincter.

The oculomotor nerve courses through the midbrain, subarachnoid space near the posterior communicating artery, and the cavernous sinus, before entering the orbit at the superior orbital fissure.

### Diagnosis

Because the nerve fibers that innervate the pupil are in the periphery of the nerve, a posterior communicating artery aneurysm commonly causes pupil dilation. When the pupil is spared, microvascular infarction due to diabetes/hypertension is more common.

Head noncontrast computed tomography (CT) should be obtained first and, if normal, can be followed by CT angiography, magnetic resonance imaging, or magnetic resonance angiography, looking for vascular lesions, demyelination, or neoplasms.

When indicated, lumbar puncture can be obtained to evaluate for ruptured aneurysm, infection, or inflammatory reaction.

## Management

Addressing and identifying the underlying cause is paramount.

Consultation and/or close outpatient follow-up with neurology and ophthalmology are often required.

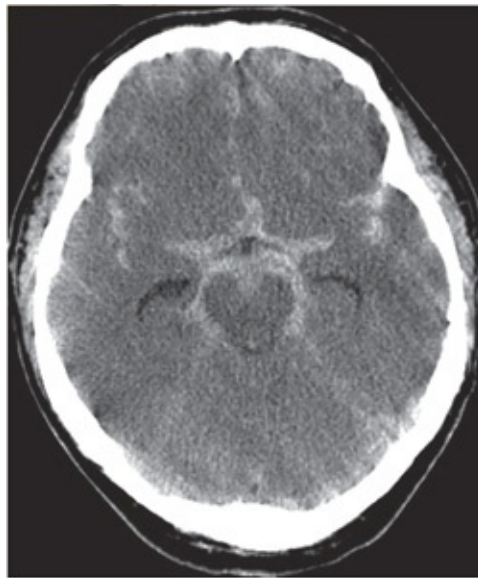
If it is an isolated nerve palsy and other more insidious causes are ruled out, “watchful waiting” is the most common treatment.

Patching and artificial tears provide symptomatic relief for persistent diplopia.



## 14. SUBARACHNOID HEMORRHAGE

Ashley Panicker, David A. Wald



**FIGURE 1.** “Star sign” of subarachnoid blood in the basal cisterns.

### Clinical Presentation

Subarachnoid hemorrhage (SAH) occurs in about 1% of patients presenting to the emergency department with headache.

In nontraumatic SAH, approximately 80% are caused by ruptured saccular aneurysms. Other causes include venous “perimesencephalic” hemorrhages (10%) and arteriovenous malformations, vascular lesions, or tumors (10%). Risk factors for SAH include smoking, hypertension, heavy alcohol use, family history of a primary relative with SAH, autosomal dominant polycystic kidney disease, and Ehlers-Danlos syndrome type IV.

The classic presentation is a sudden, severe headache typically described as the worst headache of one’s life followed by nausea, vomiting, and a transient loss of consciousness.

Physical examination may demonstrate retinal hemorrhages, decreased mental status, or focal neurologic findings. Nuchal rigidity occurs in 50% to 70% of

cases. Third nerve palsy can occur in 10% to 15% of cases.

## Diagnosis

Noncontrast head computed tomography (CT) is the initial study of choice. Sensitivity is highest in the first 6 to 12 hours (nearly 100%), 93% at 24 hours, and falls off to 50% by 1 week.

Lumbar puncture (LP) should be performed in the setting a normal noncontrast head CT. Findings indicative of SAH include xanthochromia, elevated red blood cell count that does not diminish from tube 1 to tube 4, or an elevated opening pressure.

## Management

Intubate for Glasgow Coma Scale <8, elevated intracranial pressure, hypoxemia or hypoventilation, and hemodynamic instability.

Blood pressure should be closely monitored. General recommendations are to maintain mean arterial pressure <110 mm Hg. Consider agents such as nicardipine or labetalol.

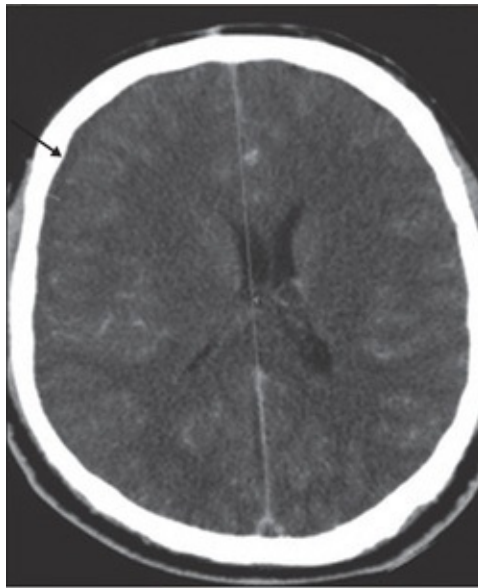
The need for routine seizure prophylaxis or antifibrinolytic agents is debated; a consensus has not been reached.

Nimodipine 60 mg orally to decrease vasospasm

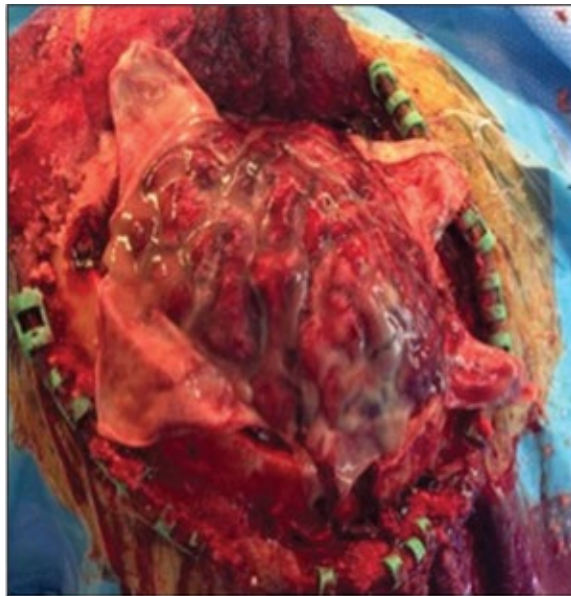
Emergent neurosurgery consultation

## 15. SUBDURAL EMPYEMA

Damali Nakitende



**FIGURE 1.** Brain CT of subdural empyema (arrow).



**FIGURE 2.** Intraoperative drainage.

Clinical Presentation

Subdural empyema is defined as a loculated infection between the dura and arachnoid matter.

This intracranial process arises from local spread of infections such as sinusitis, otitis media, or mastoiditis. It can also arise as a complication of neurosurgical procedures.

Patients may present with headache, nausea, vomiting, fevers, and a variety of neurologic symptoms depending on the location of the infection.

Infection may be complicated by cerebral venous thrombosis triggered by surrounding inflammation.

## Diagnosis

Diagnosis is guided by a combination of the history, physical exam, and imaging.

Computed tomography (CT) is typically performed as the initial imaging of choice, but magnetic resonance imaging is more sensitive.

Laboratory findings are nonspecific and generally do not alter management.

## Management

Supportive therapy may include airway management especially if the patient starts to develop changes in mental status, putting them at risk for airway compromise.

Emergent neurosurgical consultation for surgical drainage is warranted.

Antibiotics should initially be broad spectrum.

Frequent neurologic assessments are necessary, requiring management in an intensive care setting.

## 16. TROCHLEAR NERVE PALSY

Claire N. Abramoff, Michelle D. Lall



FIGURE 1. Trochlear nerve palsy of the left eye. (Images courtesy of Jason Peragallo, MD.)

### Clinical Presentation

Presenting complaint is double vision that worsens when walking down stairs, indicating difficulty with downward gaze.

Patients have binocular vertical and torsional diplopia.

Patients may have a noticeable “head tilt” to compensate for the superior oblique weakness, typically tilting to the contralateral side of the affected eye. It is most commonly idiopathic, but other etiologies include trauma, congenital defects, and vascular disease or rarely, secondary to elevated intracranial pressure.

### Diagnosis

Patients presenting with acute trochlear nerve palsy require workup to determine the etiology of the palsy, which includes complete blood count, electrolyte panel, erythrocyte sedimentation rate, C-reactive protein, rapid plasma reagin, Lyme titers, and noncontrast computed tomography of the brain.

The Parks–Bielschowsky head tilt test uses a three-step exam to assess the trochlear nerve. First, look to see if one eye is elevated compared to the other (hypertropia) in primary gaze. The superior oblique is an eye depressor so the affected eye will be elevated in trochlear nerve palsy. Next, determine if the hypertropia is greater with left or right gaze. The superior oblique muscle has

its greatest effect in adduction. Third, determine if hypertropia is greater on left or right head tilt. Hypertropia will be greatest when the affected eye is tilted down or adducted.

## Management

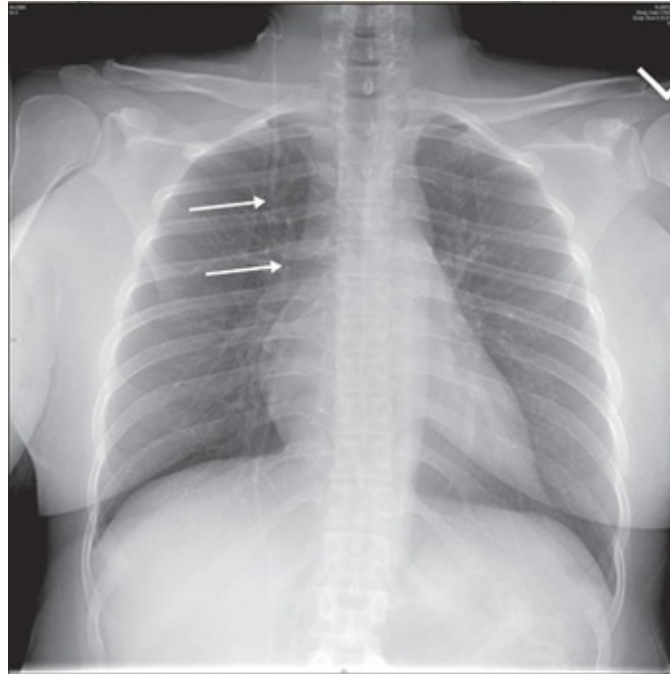
Ophthalmology referral

Traumatic and idiopathic trochlear nerve palsies will often resolve with no intervention over weeks to months. If they do not, prism therapy or strabismus surgery may be considered.



## 17. VENTRICULAR SHUNT FAILURE

Natasha Demehri, Patrick G. Meloy



**FIGURE 1.** Chest x-ray demonstrating disconnected shunt (arrows).



**FIGURE 2.** Brain CT with hydrocephalus due to malfunctioning shunt. (Image courtesy of Tarek Hanna, MD.)

## Clinical Presentation

Shunt failure may be due to choroid plexus or ependymal blockage, tube fracture, disconnection, or valve failure.

Adults typically complain of neurologic symptoms, such as headaches, irritability, vision changes, vomiting, and seizures. Geriatric patients tend to have ataxic gaits, incontinence, seizures, and lethargy.

Pediatric patients are prone to fussiness, irritability, lethargy, seizures, and vomiting. They may also develop bulging fontanelles, change in feeding habits, and high-pitched crying.

## Diagnosis

Patients may have palpable swelling along the course of the shunt.

A “shunt series,” or x-rays over the entire course of the shunt, may demonstrate a break or kink in the tubing.

Computed tomography (CT) scan of the head may demonstrate a change in ventricular size from baseline, cisternal effacement, or transependymal fluid.

A “shuntogram” involves placing a radioactive isotope in the reservoir and measuring the speed at which it traverses the tubing; delayed movement can indicate a malfunction.

## Management

Monitor for any signs of neurologic deterioration, bradycardia, or severe hypertension.

If signs of infection, fluid resuscitation and empiric antibiotics are indicated.

Neurosurgery should be consulted for a shunt-tap (obtain fluid from the shunt reservoir), admission, and frequent neurologic examinations.

Most young patients with shunts require a revision operation every 5 to 10 years as the shunt tubing degenerates causing subcutaneous granulomas, which weaken the wall of the tube.





SECTION

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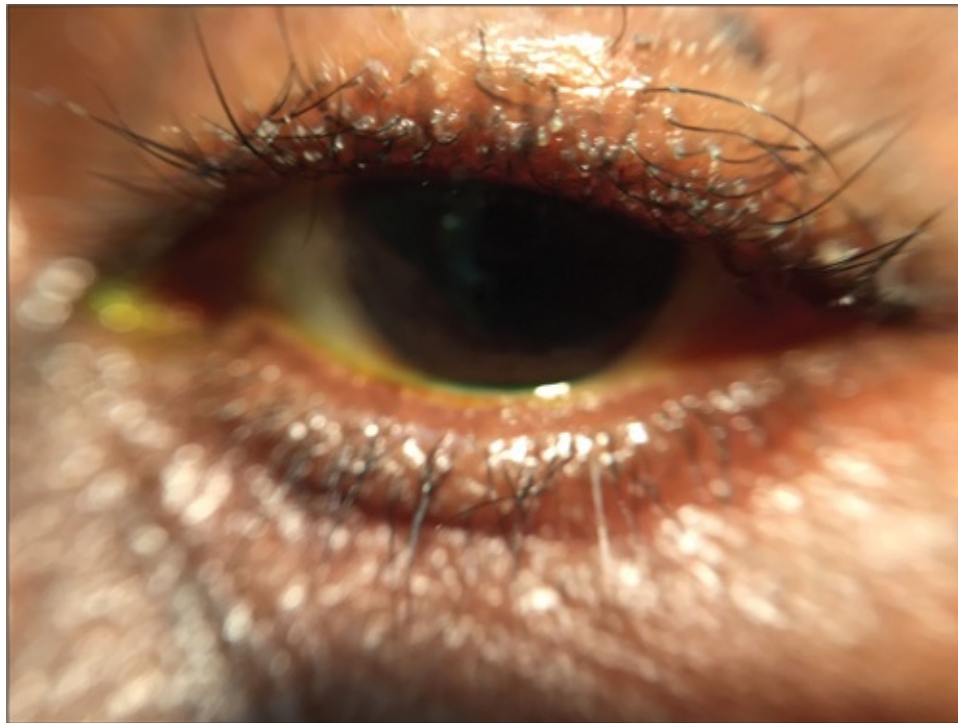
OPHTHALMOLOGIC

SECTION EDITOR

Christopher Ross

# 1. BLEPHARITIS

Timothy A. Ekhlassi, Kenya M. Williams



**FIGURE 1.** Debris seen in the eyelashes in a patient with blepharitis.

## Clinical Presentation

Commonly presents with itchy, burning eyes, often accompanied by foreign body sensation and tearing

Crusting occurs around the eyes, usually noted upon awakening.

Patients may complain of blurred vision due to poor tear film.

## Diagnosis

Exam reveals red, crusty thickened eyelids with collarettes (debris on the eyelashes), possible foamy tear film, and telangiectasias.

Patients may also have conjunctival injection, swollen eyelids, and a mild mucous discharge.

## Management

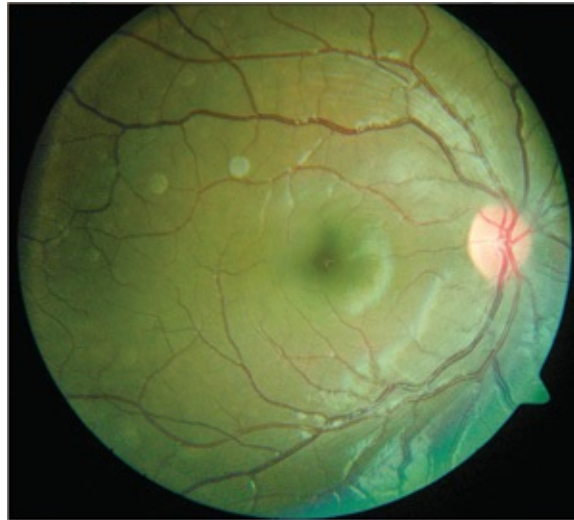
Patients should use warm compresses (e.g., a washcloth run under hot water) for 5 minutes twice a day.

After each warm compress, patients should scrub eyelid margins with a small cap full of baby shampoo on a washcloth, then re-rinse the eyelids.

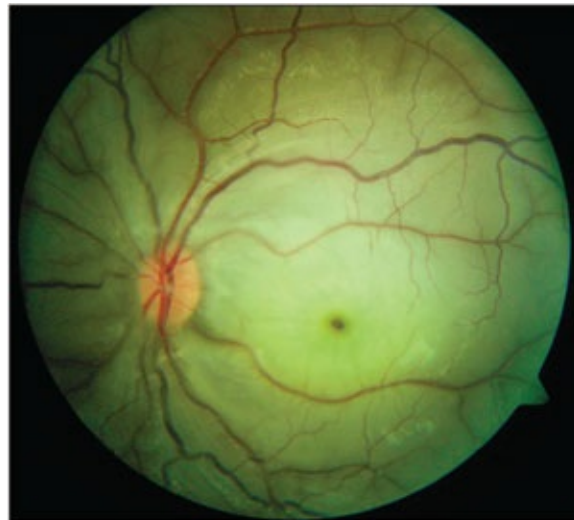
Prescribe artificial tears four times per day and, for moderate to severe blepharitis, erythromycin ointment every night at bedtime.

## 2. CENTRAL RETINAL ARTERY OCCLUSION

Matthew K. Asano



**FIGURE 1.** Normal retina, right eye.



**FIGURE 2.** Central retinal artery, left eye. Note the "cherry red spot" of the fovea and whitening of the surrounding retina.

### Clinical Presentation

Sudden, monocular, painless, and severe loss of vision

Patient may report preceding amaurosis fugax or other evidence of preceding

ischemic cerebrovascular events.

Average age of onset is seventh decade.

Atherosclerosis is thought to be cause in majority of cases.

## Diagnosis

Markedly decreased vision with afferent pupillary defect

Normal anterior eye exam with normal pressures

“Cherry red spot” of fovea due to lack of perfusion and whitening of surrounding retina

Blurring of optic disc margins

Visible emboli within vessels in small minority of cases

## Management

Prognosis for visual recovery is very poor because irreversible retinal injury occurs within 2 hours of onset.

Many treatments have been suggested, but none have proven to be consistently beneficial.

History and labs (erythrocyte sedimentation rate, C-reactive protein) to rule out giant cell arteritis is essential.

Patient will need brain, cardiac, carotid imaging, and other associated workup to rule out embolic cause.

Atypical cases (young age, lack of comorbidities) may have extensive workup and consultations.

Long-term management is cause-specific.

### 3. CENTRAL RETINAL VEIN OCCLUSION

Neerav Lamba

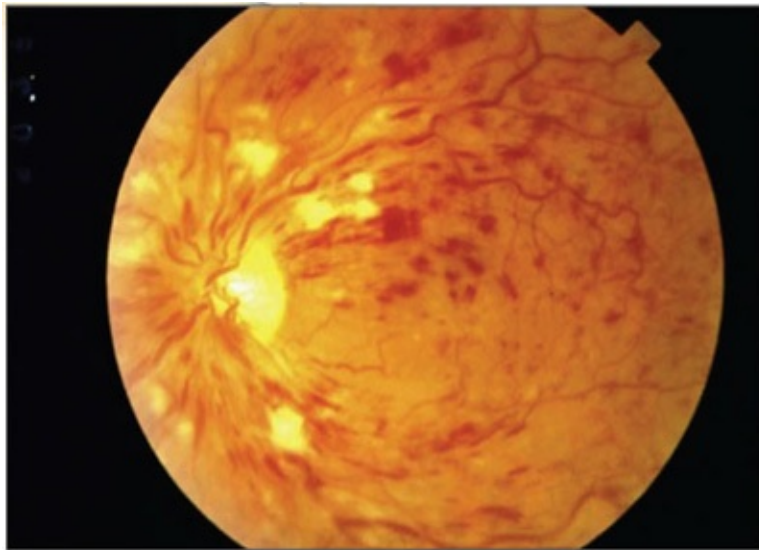


FIGURE 1. Hemorrhage and edema of the retina as seen in central retinal vein occlusion.

#### Clinical Presentation

Initially presents as painless vision loss in one eye  
Visual loss can be sudden or gradual, over a period of days to weeks.  
Visual loss ranges from mild to severe.  
Patients can present with transient obscurations of vision initially, later progressing to constant visual loss.

#### Diagnosis

If the patient has suffered from a central retinal vein occlusion, one will most likely see dilated and winding veins bulging from blood backup, bleeding into the retina, or swelling from fluid leakage when performing direct/indirect ophthalmoscopy.  
It is vital to evaluate the patient for preexisting conditions that may predispose to blood clots, such as diabetes, high blood pressure, vasculitis, high-risk



medications (e.g., oral contraceptives), and clotting disorders. Patients should undergo a complete eye examination, including visual acuity, pupillary reactions, slit lamp examination of the anterior and posterior segments, undilated examination of the iris, gonioscopy, and fundus examination with direct and/or indirect ophthalmoscope.

## Management

In older patients, laboratory testing should be directed toward identifying systemic vascular problems. In young patients, laboratory testing may be tailored depending on individual findings to include the following: complete blood cell count, glucose tolerance test, lipid profile, serum protein electrophoresis, chemistry profile, hematologic tests, syphilis serology, thrombophilic screening, activated protein C resistance, lupus anticoagulant, anticardiolipin antibodies, protein C, protein S, and antithrombin III. Medications that may increase risk of blood clots should be discontinued. Expedient consultation of an ophthalmologist can enable definitive diagnosis and ensure appropriate intervention and follow-up.

## 4. CHALAZION

Douglas Dworak

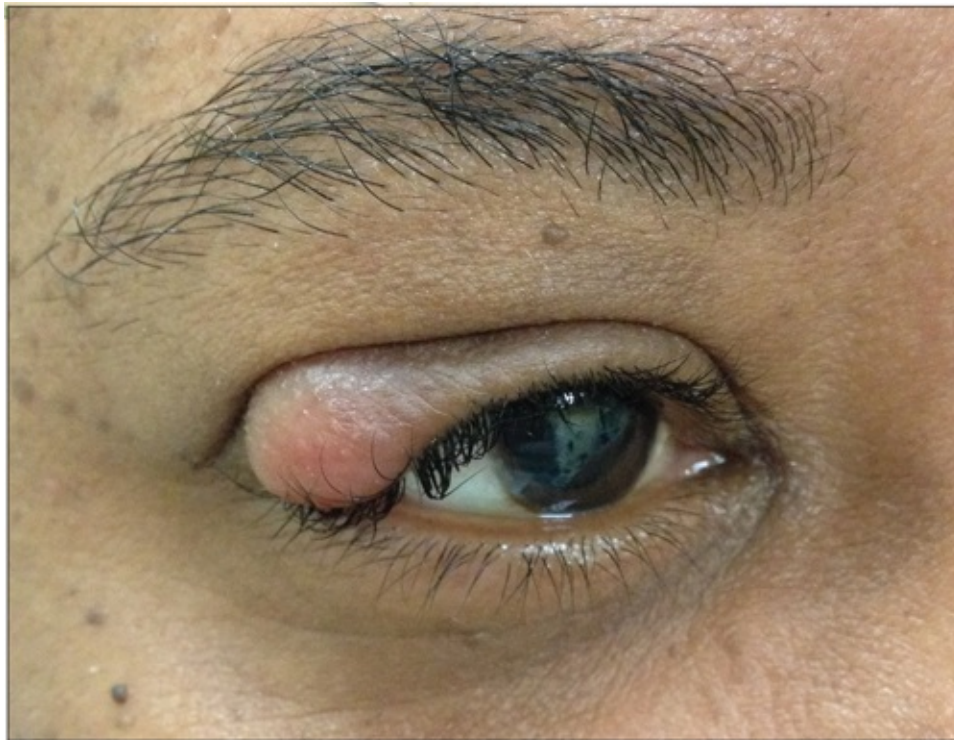


FIGURE 1. Chalazion.

### Clinical Presentation

Chronic, nontender subcutaneous eyelid nodule  
Noninfectious, inflammatory nodule secondary to blockage of the eyelid meibomian glands  
Often associated with blepharitis and acne rosacea

### Diagnosis

Palpate eyelid for a nodule and evert eyelid for examination.  
Evaluate the eyelid and glands with a slit lamp exam.  
Clinical history consistent with chronic eyelid nodule



## Management

Warm compresses four times a day and eyelid scrubs with eyelid cleanser and water.

Topical antibiotic ointment (bacitracin or erythromycin)

If refractive to conservative measures, follow up with ophthalmology for incision and curettage or intralesional steroid injection.

If recurrence despite incision and curettage, must rule out malignancy

## 5. CHEMOSIS

Timothy A. Ekhlassi, Surendar Dwarakanathan



**FIGURE 1.** A large amount of chemosis in the right eye.

### Clinical Presentation

A nonspecific finding of conjunctival irritation due to swelling or edema. Capillaries become permeable to fluid, causing the conjunctiva to have a thickened, gelatinous appearance.

Because the cornea is not covered by conjunctiva, the cornea may appear to be depressed into the globe.

### Diagnosis

Chemosis is a clinical diagnosis and its presence may indicate one of several diagnoses that can include allergic reaction, trauma, inflammation, thyroid disease (myxedema), and infection.

## Management

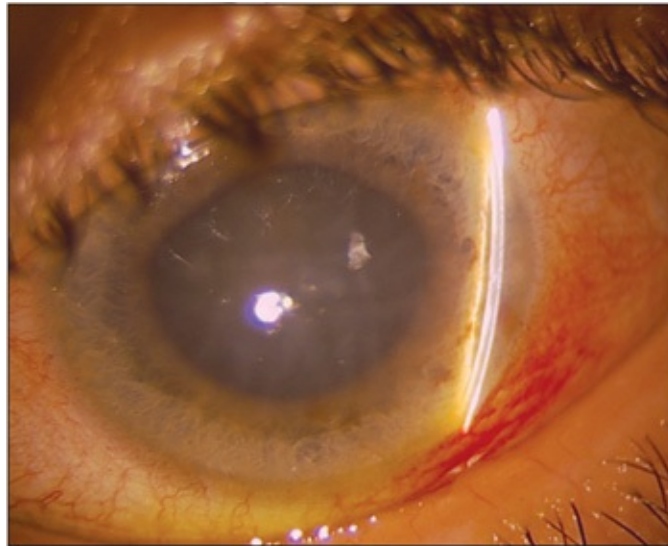
Management depends on the underlying pathophysiologic basis.

Although most commonly associated with allergy, chemosis may also be a clinical sign presenting as part of a more sinister etiology (cavernous sinus thrombosis or carotid cavernous fistula).

Allergic chemosis is treated with cool compresses, antihistamines, mast cell stabilizers, topical ocular nonsteroidal antiinflammatory drugs, and steroids (prescribed by ophthalmologists).

## 6. CLOSED-ANGLE GLAUCOMA

Matthew K. Asano



**FIGURE 1.** Primary angle closure glaucoma with shallow anterior chamber, mid-dilated pupil and cloudy cornea. (From Ehlers JP, Shah CP, Fenton GL. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*. Baltimore, MD: Lippincott Williams & Wilkins; 2008: Figure 9.4.1.)

### Clinical Presentation

Sudden onset severe eye pain with associated headache

Onset sometimes reported in dim illumination when iris is mid-dilated.

Accompanied with red eye, tearing, and photophobia

Blurring of vision may report halos around lights.

Patient can also be asymptomatic if pressure rise is slow and not reaching very high levels.

### Diagnosis

High intraocular pressure with shallow anterior chamber, injected conjunctiva

Can have corneal edema obscuring view of iris and anterior chamber

Mid-dilated fixed or sluggish pupil

Iris atrophy and pigment marks on lens can be present due to previous

episodes.

## Management

Topical intraocular pressure–lowering medications such as  $\beta$ -blockers (e.g., timolol),  $\alpha$ -agonists (e.g., brimonidine), prostaglandins (e.g., travoprost), and carbonic anhydrase inhibitors (e.g., acetazolamide).

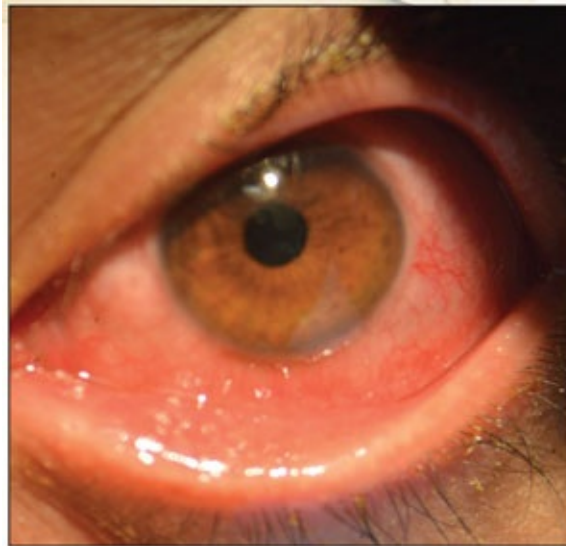
Intravenous mannitol, if renal function is normal

When pressure decreases and corneal edema clears, patient will require laser peripheral iridotomy. If laser iridotomy is not possible due to corneal clouding, surgical iridotomy would be necessary.

Patient will need evaluation of opposite eye for angle susceptible to closure attacks.

## 7. CONJUNCTIVITIS

Zachary Seagrave



**FIGURE 1.** Conjunctival injection.



**FIGURE 2.** Conjunctival papillae as seen commonly in allergic conjunctivitis.

### Clinical Presentation

Inflammation of the conjunctiva, which is the mucous membrane lining of the

eyelids and eyeball

Conjunctival hyperemia, discharge, eyelid crusting, foreign body sensation

Most common causes are infectious (bacterial or viral) and noninfectious (allergic and nonallergic).

Other causes include immunologic reactions, contact lenses use, mechanical irritation, chemicals, medications, as well as fungi.

Patients with fungal conjunctivitis are often immunocompromised.

In the United States, conjunctivitis estimated to represent nearly 1% of all emergency department visits.

## Diagnosis

Viral conjunctivitis characterized by conjunctival follicles, watery discharge, and frequently a tender, palpable preauricular lymph node. Often starts in one eye and involves the fellow eye a few days later.

Bacterial conjunctivitis characterized by conjunctival papillae and purulent discharge. Bacterial causes more often remain unilateral.

Culture in severe, recurrent, or recalcitrant cases

Allergic conjunctivitis is usually bilateral with itching, watery discharge, and conjunctival papillae; commonly seasonal and oftentimes with history or other signs of allergies.

## Management

Most cases are self-limiting, but some may progress and have sight-threatening complications.

Viral conjunctivitis is self-limited, worsening in the first 4 to 7 days, and may not resolve for 2 to 3 weeks. Supportive care with artificial tears, cool compresses, or antihistamine drops for itching.

Recommend measures to prevent spread of infectious conjunctivitis because viral conjunctivitis is highly contagious, spreading by direct contact with people or contaminated surfaces.

Treatment for allergic conjunctivitis includes eliminating the inciting agent, supportive care, topical mast cell stabilizers, and antihistamines.

Bacteria that cause conjunctivitis include *Staphylococcus aureus*,

*Streptococcus pneumoniae*, *Haemophilus influenza*, and *Moraxella catarrhalis*. Use topical antibiotic therapy such as erythromycin ointment or trimethoprim-polymyxin B drops for cases of bacterial conjunctivitis.



## 8. CORNEAL ABRASION

Erik Anderson

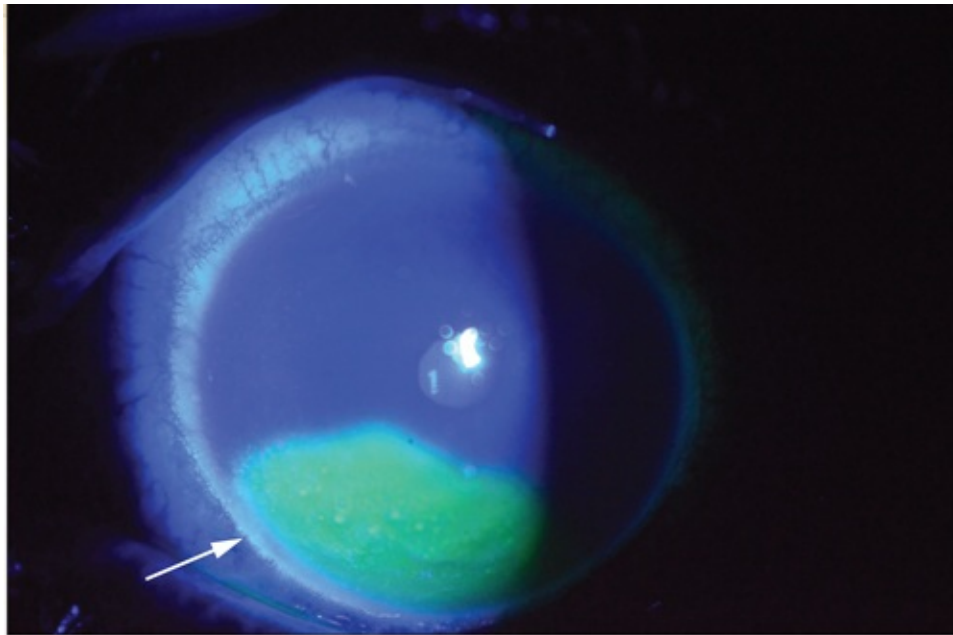


FIGURE 1. Corneal abrasion on with uptake of fluorescein (arrow).

### Clinical Presentation

One of the most frequent eye conditions presenting to emergency departments. Symptoms include pain, photophobia, tearing, foreign body sensation, and discomfort with blinking.

Pain can be severe and patients are often unable to open the affected eye.

Classified by mechanism including traumatic, foreign body, or contact lenses–related.

### Diagnosis

Epithelial defect that stains with fluorescein that may be linear scratching of the cornea to large areas of involvement.

Remember to evert the lid to look for foreign body.

Presence of an underlying or surrounding corneal opacification may indicate

an infectious etiology.

## Management

In a noncontact lens wearer, topical antibiotic ointment (erythromycin, polymyxin B/neomycin sulfate/bacitracin zinc) four times a day for 3 to 5 days.

Abrasions secondary to fingernails should be covered with a fluoroquinolone drop (ciprofloxacin 0.3%, moxifloxacin 0.5%) four times a day.

Contact lenses wearer must have antipseudomonal coverage with a fluoroquinolone topical agent.

Patching is rarely necessary and may inhibit healing.

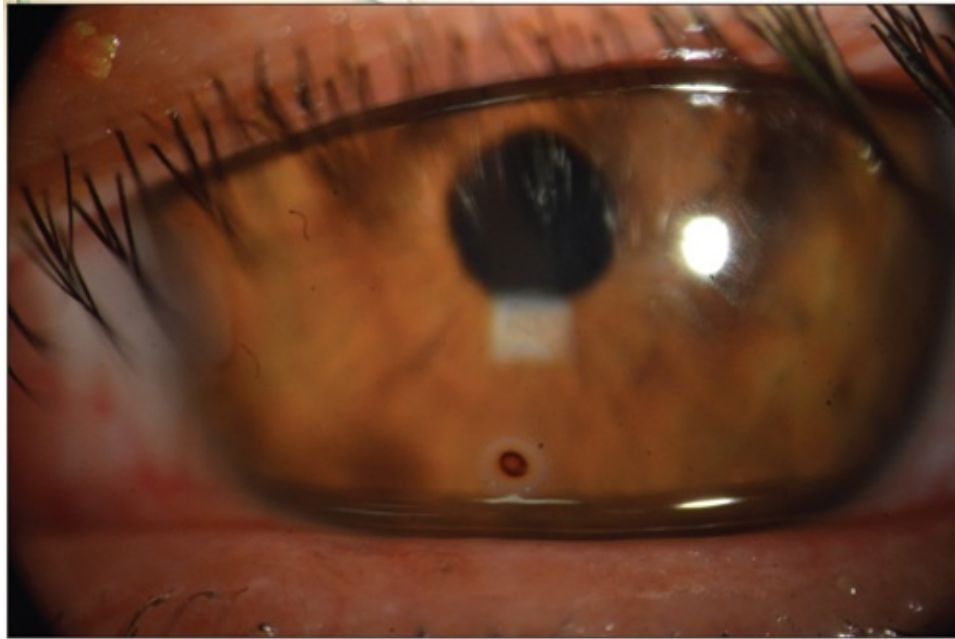
In case of an infection, DO NOT patch if the mechanism of injury involves vegetable matter, fingernails, or if the patient wears contact lenses.

Assess tetanus status.

Avoid wearing contact lenses for 10 days or until the epithelium is healed.

## 9. CORNEAL FOREIGN BODY

Erik Anderson



**FIGURE 1.** Corneal foreign body surrounded by reactive, sterile inflammatory infiltrate and rust ring.

### Clinical Presentation

Symptoms include acute onset of eye pain, foreign body sensation, tearing, and history of trauma.

Determine the mechanism of injury (metal striking metal, power tools).

A direct pathway with no safety goggle and acute onset ocular pain may suggest an intraocular foreign body.

### Diagnosis

Conjunctival injection, eyelid edema, and infiltrate may be visible surrounding the foreign body.

Diagnosis of open globe and hyphema must be excluded.

Do not forget to flip the eyelid to look for foreign body.

## Management

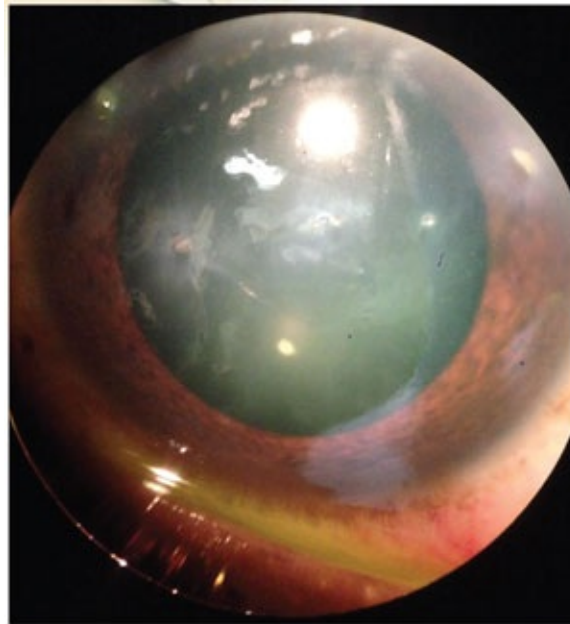
Apply topical anesthetic (proparacaine), attempt to remove foreign body first with simple cotton-tipped swab, and if unsuccessful, with a fine sterile needle. If there is a rust ring after removal of a metal foreign body, attempt to remove as much as possible if it is amenable to do so. If trained with corneal burr, then use this tool. Otherwise, remaining ring can be removed by ophthalmologist in 24 to 48 hours once the epithelium has softened.

Check tetanus vaccination status.

Treat as a corneal abrasion, and use topical antibiotics and appropriate pain control.

## **10. CORNEAL LACERATION**

Geoffrey Hill



**FIGURE 1.** Corneal laceration.

### **Clinical Presentation**

Usually due to acute trauma, patients may report a history of projectile or penetrating foreign body to the eye.

Vision is typically decreased due to anterior chamber collapse, and patients may have symptoms of tearing, photophobia, or severe pain.

Laceration may involve cornea alone or may extend through the limbus, sclera, lens, iris, or retina.

If uveal tissue is recognized on the external corneal surface or if the pupil is peaked, a penetrating laceration with uveal prolapse should be suspected.

### **Diagnosis**

Clinical diagnosis by visualization of either a full-thickness corneal defect or prolapsed uveal tissue resting on the corneal surface

For cases in which a slit lamp examination is not definitive, a Seidel test can be performed to confirm a full-thickness corneal laceration: Apply tetracaine ophthalmic drops to the ocular surface. Wet a fluorescein dye–impregnated strip with tetracaine and apply it to the area of question. Orange dye will spread over the wet areas of the normal ocular surface, but an area of brisk leakage or pooling of yellow dye over the injury site indicates a positive test. A noncontrast computed tomography of the orbit should be ordered for any patient with an open globe injury for surgical planning and to rule out intraocular foreign body.

## Management

Cover the eye with a Fox eye shield and consult an ophthalmologist.

Do not check the intraocular pressure.

Surgical repair of any open globe should be performed as soon as possible, preferably within 24 hours of presentation.

Administer broad-spectrum intravenous antibiotics and confirm up-to-date tetanus vaccination.

## **11. CORNEAL ULCER**

Neerav Lamba



FIGURE 1. Corneal ulcer.

### **Clinical Presentation**

The most common risk factors for corneal ulcers are contact lenses use and trauma.

The most common symptoms of a corneal ulcer are red eye, ocular pain, photophobia, decreased vision, and discharge.

### **Diagnosis**

Corneal ulcers present as a focal white light blocking opacity in the corneal stroma with an overlying epithelial defect that stains with fluorescein.

It is important to always culture a corneal ulcer and to include the edges and base of the ulcer, along with any discharge.

In general, corneal infections are assumed to be bacterial until proven



otherwise.

## Management

Immediately discontinue use of any contact lenses.

Treatment with topical therapy as soon as possible

Initial therapy involves cycloplegic drops for comfort and usually a potent broad-spectrum antibiotic in the fluoroquinolone class.

Fortified antibiotics should be considered for most ulcers larger than 1.5 mm, in the visual axis, or unresponsive to initial treatment.

Consider starting with a loading dose every 5 minutes for five doses and then every 30 minutes for 12 hours, then every 1 hour.

All corneal ulcers should be referred to ophthalmology for continued management.



## 12. CYTOMEGALOVIRUS RETINITIS

Kara C. LaMattina



FIGURE 1. A: Classic cytomegalovirus (CMV) retinitis. B: Perivascular CMV retinitis. C: Granular CMV retinitis.

### Clinical Presentation

Patient will have a history of HIV with CD4+ count  $<50$  or be otherwise immunosuppressed.

Patients complain of decreased vision, floaters, or scotomas.

Pain and photophobia are not features of this disease.

### Diagnosis

Slit lamp exam is diagnostic and serum testing is not indicated, although vitreal biopsies can be performed if the diagnosis is in question.

The classic form presents with areas of bleeding and retinal whitening along the major retinal vessels, described as having a “pizza pie” appearance.

The perivascular form causes frosted branch angiitis, with whitening of the retinal vessels.

The granular form causes more subtle peripheral granular opacities.

Patients often develop rhegmatogenous retinal detachments.

### Management

Patients receive intravenous ganciclovir, cidofovir, or foscarnet, with adjuvant intravitreal ganciclovir or foscarnet.

Serial fundus photographs are used to monitor the lesions for progression.

If patients are iatrogenically immunosuppressed, modifications to treatment regimen should be coordinated with their primary care physician. Retinal detachments are managed with laser demarcation or pars plana vitrectomy.

## 13. DACRYOADENITIS

Douglas Dworak

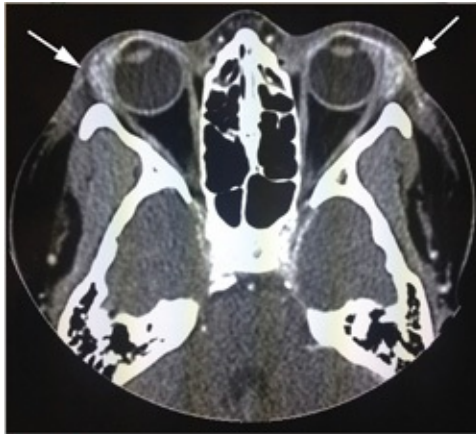


FIGURE 1. Bilateral dacryoadenitis on CT scan (arrows).



FIGURE 2. Dacryoadenitis.

### Clinical Presentation

Acute or chronic inflammation of the lacrimal gland  
Pain, erythema, and edema over the lateral aspect of the upper eyelid. Other signs include tearing, drainage, and ipsilateral preauricular lymphadenopathy.  
Typical in children and younger adults  
Commonly unilateral

Noninfectious and infectious etiologies

Viral: infectious mononucleosis, influenza, and mumps (bilateral)

Bacterial: *Staphylococcus aureus*, *Neisseria gonorrhoeae*, or streptococci

Inflammatory: sarcoidosis, nonspecific inflammatory orbitopathy (NSIO), or other lymphoproliferative diseases

## Diagnosis

Duration of symptoms and determine etiology: inflammatory, viral, or bacterial

Perform Gram stain and culture of discharge if present.

Rule out systemic involvement (complete blood count with differential, angiotensin-converting enzyme/lysozyme, fever).

Computed tomography (CT) scan of orbits to look for lacrimal gland enhancement or fluid collection and rule out bony involvement.

## Management

Etiology (inflammatory, viral, bacterial) will guide management.

Inflammatory: NSIO, treat with high-dose oral prednisone based on weight

Viral: supportive treatment with cool compresses and analgesics if needed

Bacterial: treatment guided by Gram stain and culture if available.

Amoxicillin/clavulanate or cephalexin dosed by on weight for mild to moderate conditions. If severe involvement, consider inpatient treatment.

Malignancy: bony involvement or refractive to treatment; must refer to rule out malignancy

## 14. DACRYOCYSTITIS

Douglas Dworak

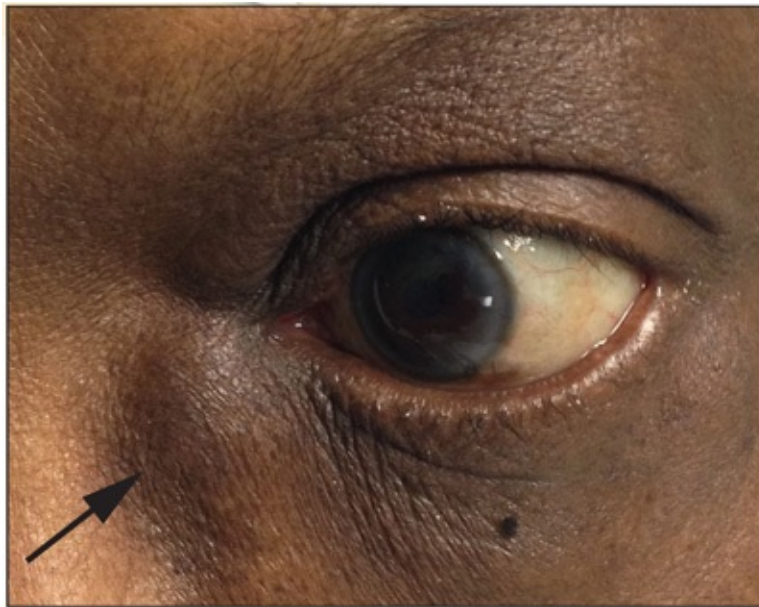


FIGURE 1. Note the swelling of the lacrimal sac medially (arrow).

### Clinical Presentation

Inflammation of the lacrimal sac

Pain, erythema, and swelling over the lacrimal sac and the medial aspect of the lower eyelid

Etiology likely related to nasolacrimal duct obstruction with a possible history of recurrence.

Muroid or purulent discharge from the punctum with digital lacrimal sac pressure

Gram-positive bacteria are most common, but consider gram-negative and atypical organisms in diabetic and immunocompromised patients.

### Diagnosis

Clinical history (previous episodes, sinus disease)

Gentle pressure with a cotton-tipped applicator to the lacrimal sac to express discharge from the punctum. Perform Gram stain and culture (if blood is present must rule out malignancy).

Computed tomography scan of orbits/sinuses in severe or refractive cases to rule out deeper associated infections such as orbital cellulitis or sinus disease

## Management

Systemic antibiotics are required.

Children

Afebrile, stable: oral amoxicillin/clavulanate or cefpodoxime

Febrile, unstable, unreliable: inpatient treatment with intravenous cefuroxime

Adults

Afebrile, stable: oral cephalexin or amoxicillin/clavulanate

Febrile, unstable, and unreliable: inpatient treatment with intravenous cefazolin

Warm compresses four times a day and gentle digital massage to area

Topical antibiotics can be added in addition to systemic medications.

If abscess develops, consider incision and drainage and surgical interventions for chronic cases.



## 15. HERPES SIMPLEX KERATITIS

Matthew K. Asano

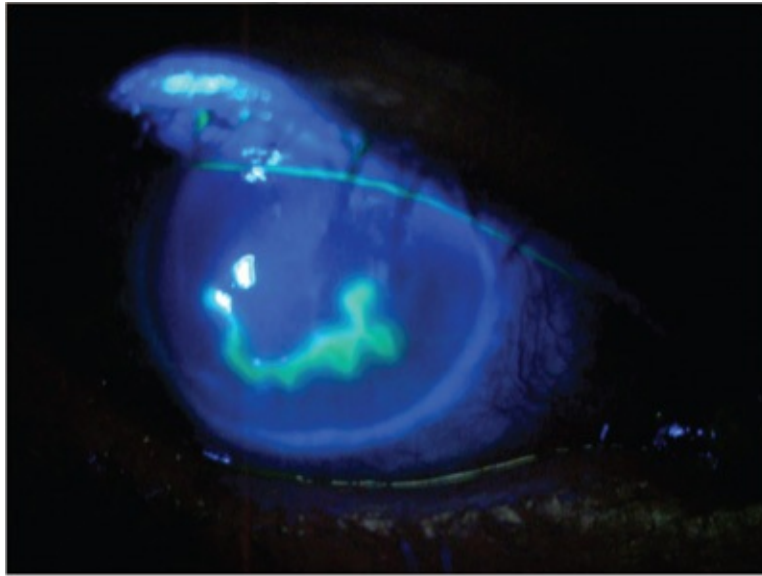


FIGURE 1. Typical HSV dendrite viewed with fluorescein staining and blue light.

### Clinical Presentation

Nearly all ages can be affected by herpes simplex virus (HSV) keratitis. Patients usually complain of unilateral red eye with tearing, photophobia, visual blurring, and watery discharge for several days. Patients may also complain of a foreign body sensation. Patient may have history of oral or genital ulcers indicative of HSV-1 or HSV-2 infection.

### Diagnosis

Diagnosis based on history and physical examination findings. Exam of cornea with fluorescein staining of epithelial dendritic lesion(s) is usually diagnostic. Dendrites may be singular or multiple and vary in length from smaller than a millimeter to several millimeters. An associated ciliary flush and decreased corneal sensation may be identified.

along with a haze or scarring of the deeper corneal stroma.  
Intraocular pressure may be elevated as a response to the virus.  
Culture large or atypical lesions for possible bacterial infection.  
Large ulcers or late presentations with scarring and vascularization can be diagnostic challenges.

## Management

Topical antiviral eye drops (e.g., trifluridine, ganciclovir, acyclovir, and vidarabine) for 7 to 10 days

Topical corticosteroids should *never* be given for acute infection but may be used by ophthalmology for deeper stromal inflammation after the epithelial defect is healed and the active infection has resolved.

Long-term oral acyclovir may be required for recurrent disease.

Corneal transplant may be necessary if scarring affects vision.



## 16. HERPES ZOSTER OPHTHALMICUS

Kara C. LaMattina



FIGURE 1. Herpes zoster involving the V1 dermatome.



FIGURE 2. Hutchinson's sign.

**Clinical Presentation**

Patients are generally >60 years old or otherwise immunocompromised. Presenting symptoms include pain, tingling, and rash in the V1 distribution. Patients may complain of decreased visual acuity, redness, floaters, scotomas, and foreign body sensation.

## Diagnosis

Slit lamp exam is diagnostic.

Patients will have vesicular dermatomal rash along V1 distribution; a lesion at the tip of the nose (Hutchinson's sign) is highly correlated with ocular involvement.

Patients may have conjunctivitis or pseudodendrites (raised dendritiform lesions without terminal bulbs).

Commonly develop uveitis associated with elevated intraocular pressure  
May also develop scleritis, retinitis, choroiditis, and optic neuritis

## Management

If patient is <40 years old and not immunocompromised, workup is indicated. Oral acyclovir 800 mg five times daily or valacyclovir 1 g three times daily for 7 to 10 days

Patients with conjunctival or corneal involvement are treated with erythromycin ointment four times daily and artificial tears every 1 to 2 hours (topical antivirals are generally not indicated in the management of zoster).

If stromal keratitis or uveitis is present, topical steroids are indicated.

If the posterior segment is involved, intravenous or intravitreal antivirals are indicated.

## 17. HORDEOLUM

Douglas Dworak

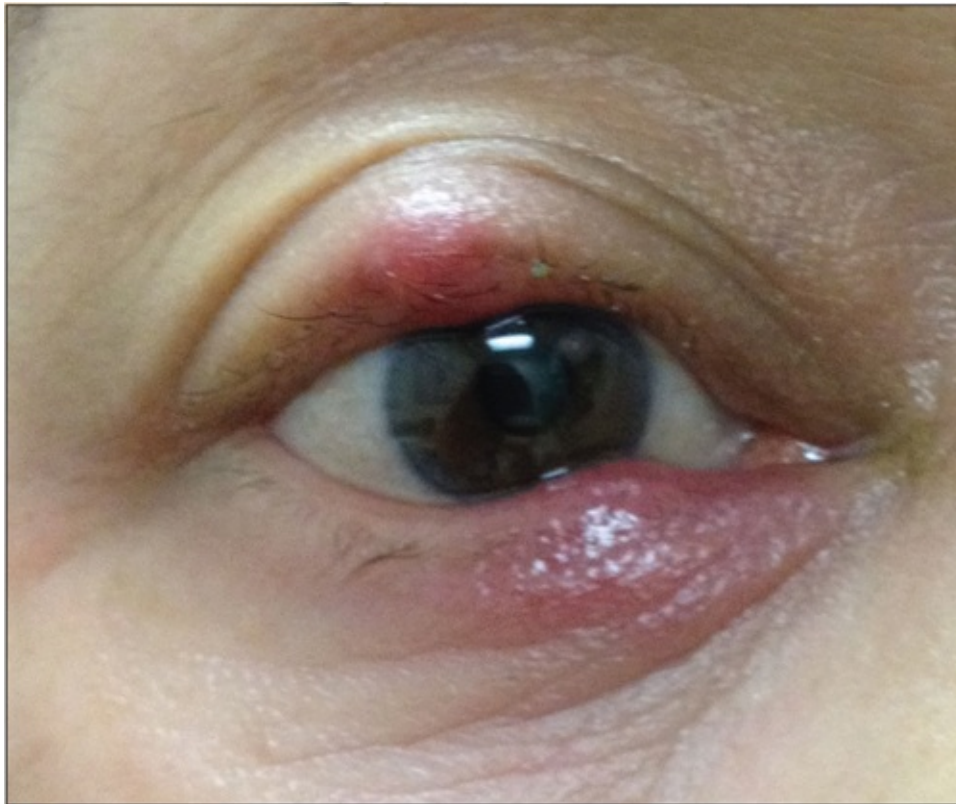


FIGURE 1. Hordeolum.

### Clinical Presentation

Acute, eyelid swelling and erythema with a localized raised cutaneous nodule  
Infection of the internal or external glands of the eyelid (glands of Zeis and meibomian glands) around eyelash follicle  
Commonly caused by *Staphylococcus* species  
Can potentially progress to preseptal and septal cellulitis

### Diagnosis

Palpate eyelid for a nodule and evert eyelid for examination.

Evaluate the eyelid and glands with a slit lamp exam.  
Clinical history consistent with acute eyelid swelling

## Management

Warm compresses four times a day and gentle digital massage  
Topical antibiotic ointment (bacitracin or erythromycin) for associated drainage or blepharitis  
Consider systemic oral antibiotics (doxycycline, cephalexin) if moderate to severe or suspect preseptal or septal cellulitis.  
Incision and curettage if lesion worsens or does not respond to conservative treatment

## **18. HYPHEMA**

Kara C. LaMattina



**FIGURE 1.** Layered hyphema.

### **Clinical Presentation**

The most common etiology is blunt trauma.

Symptoms may include pain and decreased vision.

The patient presents with blood in the anterior chamber, which may be layered, dispersed, or completely fill the anterior chamber.

### **Diagnosis**

Slit lamp exam is diagnostic.

A layered hyphema presents as red blood cells settled in the inferior part of the anterior chamber with a clear demarcation line.

A dispersed hyphema may be seen as pigmented cells in the anterior chamber.

An “eight-ball” hyphema presents as a black clot filling the entire anterior chamber.

It is important to check intraocular pressure because extremely elevated pressures may require surgical intervention.

## Management

A metal or plastic shield should be taped over the eye at all times, and activity restricted.

Pain management with acetaminophen (rather than aspirin or nonsteroidal antiinflammatory drugs)

Sickle cell testing should be performed because these patients have higher risk of intraocular pressure (IOP) spikes and certain medications are contraindicated.

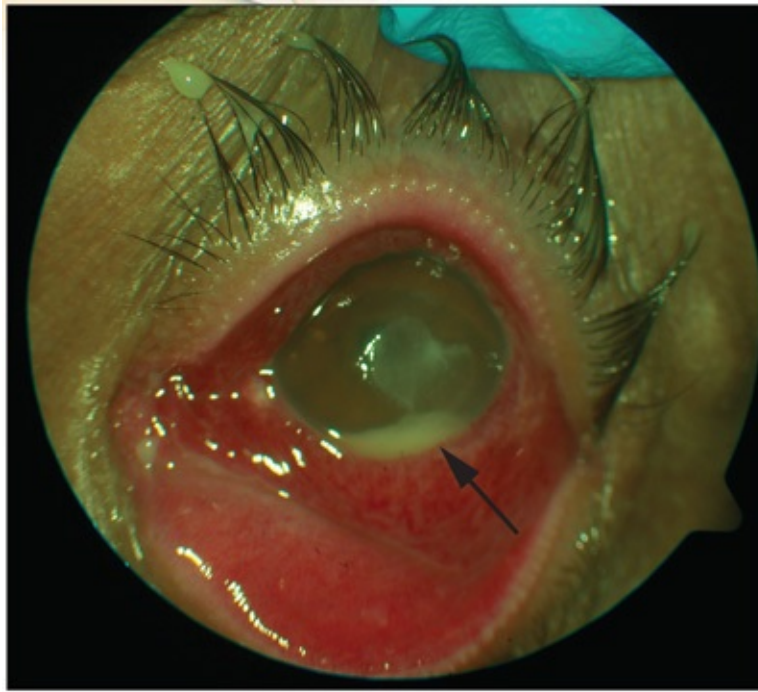
Daily follow-up with ophthalmologist for 5 days to monitor for rebleeds

In cases of extreme IOP elevation or prolonged moderate elevation, anterior chamber washout may be indicated.



## 19. HYPOPYON

Neerav Lamba



**FIGURE 1.** Layering at the bottom of the anterior chamber (arrow).

### Clinical Presentation

A hypopyon is leukocytic exudate seen as a white substance sitting at the bottom of the anterior chamber in the eye.

It can be clearly visualized as a fluid level, and the sedimentation is governed by gravity.

A hypopyon is usually accompanied by redness of the conjunctiva and the underlying episclera.

### Diagnosis

Causes of a hypopyon include infection, cellular immune reactions influenced by patterns of human leukocyte antigen expression, systemic inflammatory conditions, and trauma.

A careful slit lamp exam is necessary to evaluate the characteristics of the hypopyon (size, shape, color) and for any other ocular lesions. It is important to illicit a careful patient medical history which can often determine the presence of associated risk factors or systemic conditions.

## Management

In treating a hypopyon, it is essential to diagnose the cause of the inflammatory reaction and treat the patient accordingly. Immediate referral to an ophthalmologist should occur because a hypopyon can be associated with infectious keratitis or endophthalmitis.



## 20. LENS DISLOCATION

Geoffrey Hill



FIGURE 1. Lens subluxation.

### Clinical Presentation

In cases of Marfan syndrome, homocystinuria, or other connective tissue disease, onset can be spontaneous, but lens dislocation is more often related to blunt head trauma or direct ocular trauma.

Patients may report a painless but dramatic, sudden-onset decrease in visual acuity or classically monocular diplopia.

### Diagnosis

Upon slit lamp examination, the crystalline lens—normally located directly behind the iris—is absent.

Visual acuity is typically in the counting fingers range but may improve significantly with pinhole testing.

## Management

Lens dislocation is not an emergency but does require surgical intervention for repair. Routine outpatient referral to an ophthalmologist is appropriate.

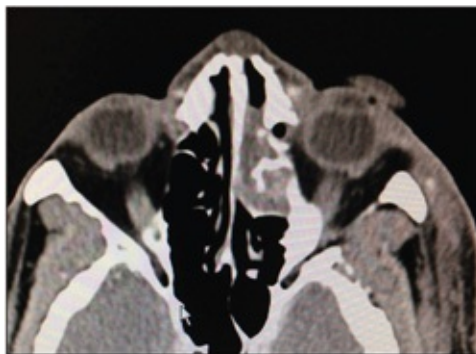
## 21. ORBITAL CELLULITIS



Paul O. Phelps



**FIGURE 1.** Orbital cellulitis of the left eye. Accompanying video of same patient demonstrates restrictions in eye movements.



**FIGURE 2.** CT scan demonstrating ethmoid sinusitis and adjacent orbital inflammation.

### Clinical Presentation

Patients present with eye pain, decreased vision, conjunctival hyperemia, and loss of eye motility.

Eyelid edema, erythema, and tenderness are also often present.

An afferent pupillary defect and diminished color vision may indicate optic nerve involvement.

Onset is usually rapid, and clinical suspicion should be increased in children.

Orbital cellulitis can be distinguished from preseptal cellulitis by ophthalmoplegia, pain with eye movements, and proptosis as well as imaging studies.

Severe orbital inflammation should be presumed to be orbital cellulitis until proven otherwise.

## Diagnosis

Diagnosis is made by clinical suspicion and imaging studies.

Leukocytosis and fever are often present.

A computed tomography (CT) or magnetic resonance imaging of the orbits often demonstrates concomitant sinusitis and is generally required to diagnose orbital cellulitis.

## Management

Intravenous antibiotics that focus on staphylococcal and streptococcal involvement should be administered promptly when orbital cellulitis is suspected.

Incision and drainage may be required in cases with associated abscesses.

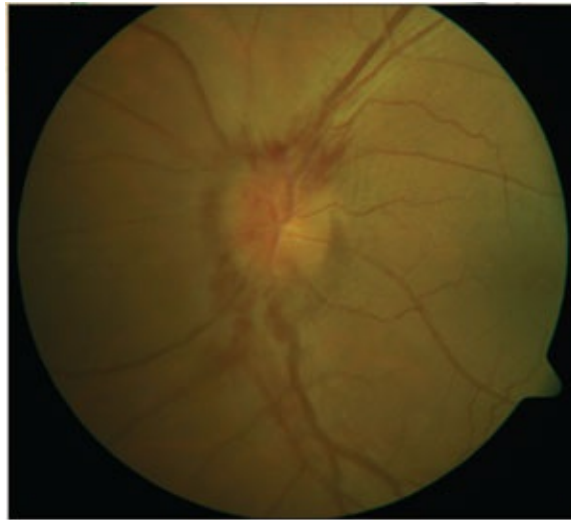
Drainage may relieve pain and optic nerve compression.

An ophthalmologist should be consulted to determine the vision and the extent of optic nerve involvement.

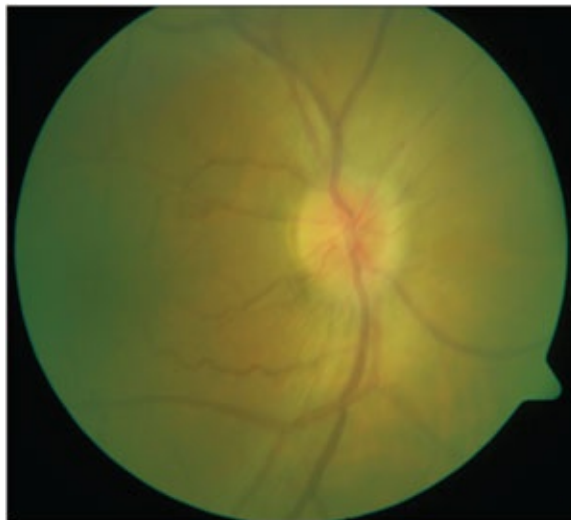
Symptoms should decrease with the administration of antibiotics, but nonspecific orbital inflammation may be suspected if no clinical improvement is noted within 24 to 48 hours.

## 22. PAPILLEDEMA

Zachary Seagrave



**FIGURE 1.** Severe papilledema demonstrating hyperemia and swelling of the optic disc with associated flame hemorrhages.



**FIGURE 2.** Papilledema with elevation of the optic disc and blurring of the disc margins.

### Clinical Presentation

Papilledema is characterized by optic disc edema in the setting of increased intracranial pressure resulting from a variety of conditions including an

intracranial mass, hydrocephalus, cerebral edema, increased cerebrospinal fluid (CSF) production, decreased CSF absorption, obstruction of venous outflow, and idiopathic intracranial hypertension.

Symptoms of increased intracranial pressure including headache, nausea, and vomiting

May have transient visual obscurations—episodes of vision loss lasting seconds with full recovery of vision, often associated with orthostatic changes.

Often associated with orthostatic changes

Untreated, chronic papilledema can lead to progressive visual field loss, starting with loss of peripheral vision that can progress in rare case to total loss of vision.

## Diagnosis

Visual acuity is usually normal, and pupillary responses are normal.

The earliest finding of papilledema is loss of spontaneous venous pulsations (20% of general population does not have detectable venous pulsations).

Fundus examination demonstrating bilateral hyperemia and swelling with elevation of the optic disc, typically seen as blurring of the disc margin. Flame hemorrhages and cotton wool spots can also appear.

Consider laboratory evaluation (erythrocyte sedimentation rate/C-reactive protein/complete blood count) if ischemic optic neuropathy from giant cell arteritis/temporal arteritis is suspected.

## Management

Urgent neuroimaging, magnetic resonance imaging (MRI) with gadolinium with magnetic resonance venogram of the head is preferred or computed tomography scan if MRI not immediately available.

Lumbar puncture with CSF analysis and opening pressure measurement if neuroimaging does not reveal a mass lesion, venous thrombosis, or hydrocephalus

Treatment directed at the underlying cause of the increased intracranial pressure



## **23. PERIORBITAL (PRESEPTAL) CELLULITIS**

Timothy A. Ekhlassi, Kenya M. Williams



**FIGURE 1.** Periorbital cellulitis of the right eye.

### **Clinical Presentation**

Patients complain of pain, redness, and swelling of the eyelid and periorbital area.

Patient often has a history of localized infection or inflammation, including sinusitis, abrasions, hordeolum, or insect bites.

Patient may complain of mild fever.

### **Diagnosis**

Pertinent positives include tense, edematous eyelids, erythema, warmth, and

tenderness. Edematous eyelids may make examination difficult. Pertinent negatives include no proptosis, no restriction of eye movement, ophthalmoplegia, and no pain with eye movement because these may be indicative of a deeper infection. Orbital cellulitis must be ruled out. Orbits computed tomography can help differentiate between preseptal and orbital cellulitis.

## Management

Mild cases in patients older than 5 years can be treated with oral broad-spectrum antibiotics on an outpatient basis for a minimum of 10 days. Special care should be taken to tailor antibiotic therapy when methicillin-resistant *S. aureus* exposure is suspected.

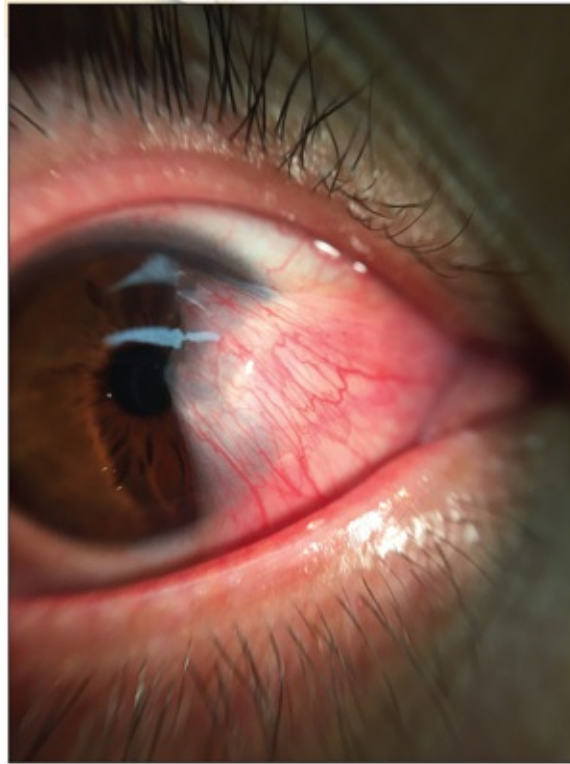
Warm compresses may help to speed clearance. If an identifiable skin lesion is present, topical antibiotic ointment such as erythromycin, bacitracin, or triple antibiotic ophthalmic ointment may be applied.

Severe cases, children younger than 5 years, and patients who appear toxic should be admitted for intravenous antibiotic therapy.



## 24. PTERYGIUM

Geoffrey Hill



**FIGURE 1.** Triangular white growth over the cornea consistent with a pterygium.

### Clinical Presentation

Degenerative, slowly progressing disorder of the conjunctiva  
Common in those with Hispanic, Middle Eastern, or Mediterranean ethnicity  
Risk factors are ocular surface dryness and ultraviolet exposure.  
Patients may be asymptomatic or may report chronic, mild to moderate tearing, foreign body sensation, redness, or irritation.

### Diagnosis

Slow-growing, chronic in onset  
Appears as wing-shaped or triangular, fleshy, white growth of conjunctiva

extending onto the cornea

Conjunctiva may be focally or diffusely injected.

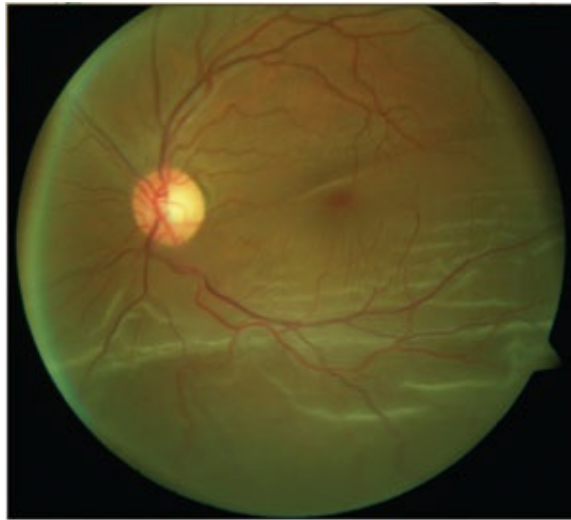
## Management

Recommend frequent artificial tears for lubrication and sunglasses for ultraviolet light protection.

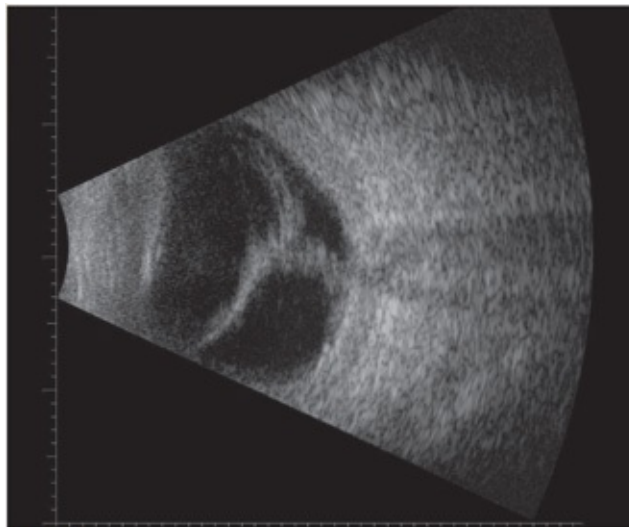
Referral to ophthalmologist for surgical removal if extensive corneal involvement or if visual acuity is affected by overgrowth onto visual field

## 25. RETINAL DETACHMENT

Paul O. Phelps



**FIGURE 1.** Rhegmatogenous retinal detachment involving the macula that demonstrates corrugated and opaque retina.



**FIGURE 2.** B-scan ultrasound demonstrating a funnel-shaped total retinal detachment.

### Clinical Presentation

Patient may present up to 1 year after traumatic event.  
Often, patients have no history of trauma but high myopia (i.e.,

nearsightedness).

Almost all patients experience flashes and floaters, with variable vision loss that may range from a nuisance to visually disabling.

## Diagnosis

A dilated fundus examination by an ophthalmologist is generally required for accurate diagnosis.

Patients may have vision loss with symptoms of a curtain coming down over their vision and/or loss of visual fields.

Ultrasound may be helpful in the emergent setting to visualize the detachment.

## Management

Emergent referral to an ophthalmologist is recommended in patients with a clinical presentation that suggests retinal detachment. Treatment may range from reassurance and education to scleral buckle or vitrectomy.

Patients with good vision and retinal detachment are more likely to have a macula-sparing retinal detachment, which is best repaired urgently.

## 26. RUPTURED GLOBE

Paul O. Phelps



FIGURE 1. Left eye demonstrating corneal and scleral lacerations with protruding uveal tissue.

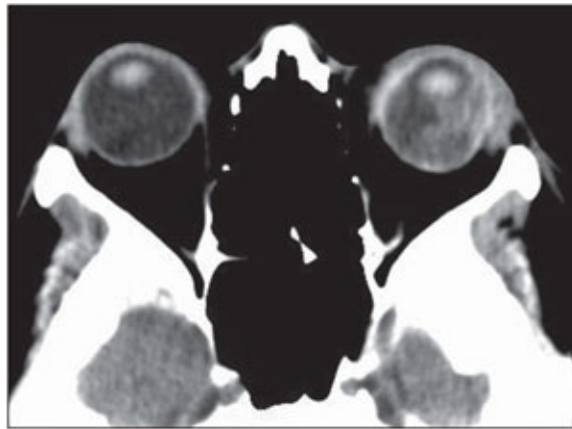


FIGURE 2. CT scan demonstrating globe rupture on the left.

### Clinical Presentation

Ruptured globe is usually caused by direct injury to the eye. Blunt trauma usually causes a rupture at the site of greatest structural weakness, which is near the equator and directly behind the rectus muscles. This area is not visible to direct inspection. Patients typically present soon after blunt or penetrating trauma complaining of pain and visual impairment or loss.

Glass is especially likely to cause deeper tissue injury and globe involvement. The patient's ultimate vision (after repair) is likely to mirror the patient's vision on presentation.

## Diagnosis

Signs of ruptured globe include deflated globe and protruding iris, choroid, or other internal tissues.

An irregular pupil is another common sign of a ruptured globe.

The eye may be stained with fluorescein to check for leaking aqueous humor (i.e., Seidel's sign).

A 360° subconjunctival hemorrhage may indicate a scleral laceration.

Orbits computed tomography (CT) should be ordered to determine if a retained foreign body is present prior to surgery.

## Management

The patient's eye should be covered with a fox-shield (or other hard cover) when an open globe is suspected. Checking the intraocular pressure with a Tono-Pen should be deferred.

Surgical treatment is required urgently to close the globe and prevent further tissue loss.

An ophthalmologist should be consulted emergently when a ruptured globe is suspected.



## 27. SUBCONJUNCTIVAL HEMORRHAGE

Erik Anderson



**FIGURE 1.** A 360° subconjunctival hemorrhage secondary to blunt trauma with a ruptured globe (posterior scleral laceration).



**FIGURE 2.** Simple subconjunctival hemorrhage likely secondary to hypertension or valsalva.

### Clinical Presentation

Usually asymptomatic with a red eye in one quadrant  
Hemorrhage in all quadrants can indicate a ruptured globe.

## Diagnosis

Determine etiology: Valsalva maneuver (heavy lifting, coughing, choking, constipation, vomiting), traumatic (eye rubbing), hypertension, bleeding disorder, antiplatelet or anticoagulant, and idiopathic.

## Management

A subconjunctival hemorrhage is typically a self-limiting condition that will clear in 2 to 4 weeks without treatment. Artificial tears can be used two to three times daily until the hemorrhage has cleared.

Reassurance is usually all that is required.

If recurrent, check coagulation studies and refer to ophthalmology to rule out a conjunctival lesion.

In traumatic cases, rule out an open globe with a CT scan of the orbits.



## 28. TRAUMATIC IRITIS

Zachary Seagrave

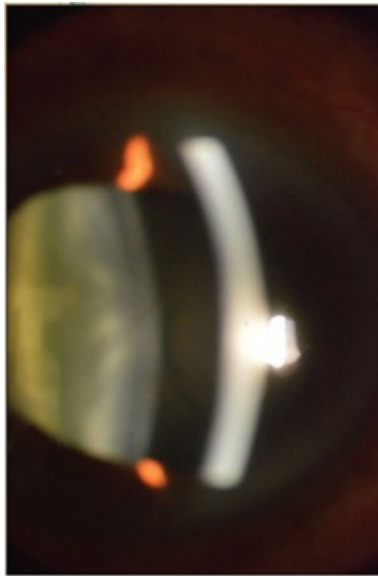


FIGURE 1. Traumatic iritis with ciliary flush.

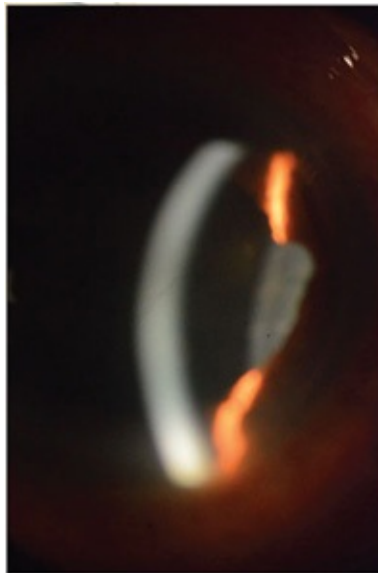


FIGURE 2. Traumatic iritis with associated sphincter tear.



FIGURE 3. Cell and flare.

## Clinical Presentation

Dull, aching, or throbbing eye pain

Redness, tearing, and photophobia

Variable visual acuity but usually decreased vision is endorsed.

History of eye trauma typically within 3 days of symptom onset

Nontraumatic causes of iritis include a wide variety of infectious, inflammatory, and infiltrative processes such as Reiter syndrome, tuberculosis, sarcoid, and syphilis.

## Diagnosis

The cardinal sign of iritis is a ciliary flush, which gives the appearance of red ring circumscribing the iris.

Ophthalmic examination including visual acuity, pupillary responses, intraocular pressure measurement, slit lamp examination, and fundus examination

White blood cells and flare in the anterior chamber visualized at slit lamp under high magnification while focusing into the anterior chamber.

May have poorly reactive pupil in the traumatized eye

## Management

Cycloplegic medication (e.g., atropine 1% twice a day, homatropine three times a day, cyclopentolate 2% three times a day, or scopolamine 0.25% twice a day)

Consider topical steroid drop (e.g., prednisolone acetate 1% four times a day) if a corneal epithelial defect is not present under direct consultation with ophthalmology.

Follow up with ophthalmologist.



SECTION

D

EAR, NOSE, THROAT

SECTION EDITOR

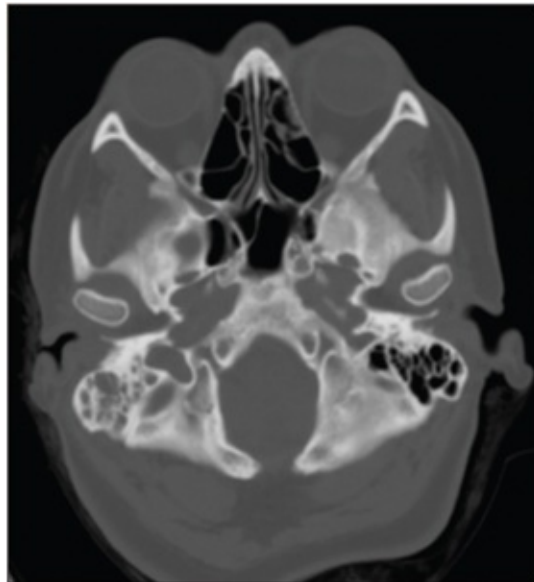
Christopher Ross

# 1. ACUTE MASTOIDITIS

Christopher Ross, Damali Nakitende



**FIGURE 1.** Pinna proptosis and mastoid swelling in a patient with mastoiditis.



**FIGURE 2.** Acute mastoiditis (*right*) with opacification and some erosion of the mastoid air cells.

Clinical Presentation

Mastoiditis typically arises when infection or inflammation spreads from the middle ear to the air cells, inducing destruction of the mastoid bone and periosteum.

Local extension from this inflammatory process can result in meningitis, intracranial abscess, cavernous sinus thrombosis, and facial nerve palsy. Patients commonly present with fevers, chills, postauricular pain, and discharge from the external auditory canal.

Children may only present with only irritability and decreased appetite. Risk factors for this infection include diabetes, immunocompromised, recurrent acute otitis media, and presence of a cholesteatoma.

## Diagnosis

Guided by a combination of the history, physical exam, and imaging. Physical exam may show a bulging or perforated erythematous tympanic membrane; purulent discharge in the external auditory canal; proptosis of the pinna; and pain, erythema, and swelling of the mastoid process.

Computed tomography of the head or mastoid will show fluid-filled air cells confirming clinical suspicion of disease process.

Laboratory findings rarely change management. However, blood cultures should be obtained if the patient is febrile.

## Management

Early consultation of an otolaryngologist is recommended for acute mastoiditis due to the severity of the potential complications.

Most cases are admitted for treatment with parenteral antibiotic therapy with penicillins, cephalosporins, or vancomycin.

This is followed by a course of oral antibiotics once there is clinical improvement and physical signs have diminished.

Antibiotics should provide coverage for common bacteria such as *Streptococcus*, *Pseudomonas*, *Staphylococcus aureus*, *Moraxella catarrhalis*, and *Haemophilus influenzae*.

Surgical management with irrigation and debridement is reserved for

refractory cases.



## 2. ANGIOEDEMA

Daniel Stein, Bory Kea



FIGURE 1. Sublingual angioedema.

### Clinical Presentation

Angioedema is characterized by local swelling of the skin and/or mucosal membranes due to “leaky” capillaries and subsequent extravasation of fluid into the interstitium.

It most typically involves surfaces that have loose connective tissue: lips, mouth, tongue, throat, larynx, and uvula.

Angiotensin-converting enzyme (ACE) inhibitor–induced angioedema affects roughly 0.2% to 0.7% of patients on an ACE inhibitor.

Hereditary angioedema is a rare autosomal dominant genetic disorder that accounts for roughly 2% of all cases of angioedema and is caused by a deficiency in functional C1 esterase inhibitor. Triggers can include minor trauma, cold exposure, stress, and viral illnesses.

### Diagnosis

Diagnosis of angioedema is based on history and physical examination. There are no confirmatory laboratory studies or imaging modalities that are



available in the emergency department that confirm the diagnosis. Hereditary angioedema can be diagnosed in the outpatient setting using serum complement factor 4 (C4) levels, C1 esterase inhibitor levels, and C1 esterase inhibitor functional levels (if available).

## Management

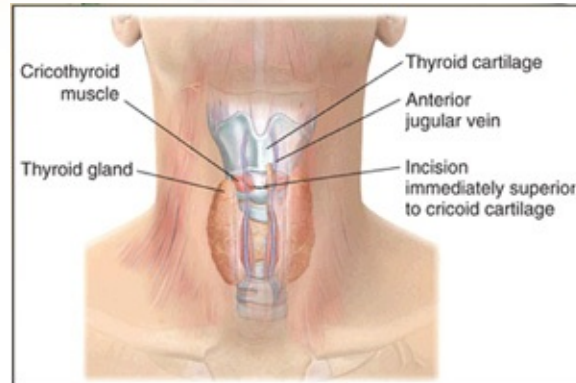
Treatment of angioedema in the emergency department is supportive, with early aggressive interventions if needed for airway management.

Medications used to treat typical allergic reactions such as corticosteroids and antihistamines are often used but generally are not beneficial because angioedema is not associated with an increase in immunoglobulin E. There has been no clear benefit shown in using epinephrine.

Patients with an acute exacerbation of hereditary angioedema should ideally be managed in coordination with an appropriate specialist. Treatment options include C1 esterase inhibitor replacement, ecallantide (a kallikrein inhibitor), icatibant (a bradykinin receptor antagonist), and fresh frozen plasma.

### 3. CRICOTHYROIDOTOMY

Daniel Stein, Bory Kea



**FIGURE 1.** Anatomy of the neck for performing a cricothyroidotomy. (From Britt LD, Peitzman A, Barie P, et al. *Acute Care Surgery*. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)



**FIGURE 2.** Performing a cricothyroidotomy using a bougie.

#### Clinical Presentation

Rates of failed emergency department (ED) intubations requiring cricothyroidotomy or other surgical airway management is <0.6%. The success rate of a cricothyroidotomy ranges from 96% to 100%. When compared to a tracheostomy, a cricothyroidotomy is easier, faster, and safer because the superficial landmarks can typically be seen and palpated.

#### Diagnosis

A cricothyroidotomy is a procedure performed in emergent conditions on

patients in respiratory failure in whom orotracheal or nasotracheal intubation is not possible.

There are few contraindications to a cricothyroidotomy. If there is injury to the larynx, thyroid cartilage, or cricoid cartilage, a tracheostomy is preferable.

Children younger than 10 to 12 years old should have a needle cricothyroidotomy performed.

## Management

There are multiple techniques described for the ED cricothyroidotomy. We will describe the traditional horizontal incision:

The larynx must first be stabilized with the thumb and middle finger of the operator's nondominant hand. The index finger should palpate the cricothyroid membrane.

An 11 scalpel blade should be used to make a stab incision horizontally, about 1.5 cm in the midline through the cricothyroid membrane. Do not remove the scalpel.

Insert the tracheal hook along the cephalad face of the scalpel. Engage the inferior lip of the tracheal hook and lift it anteriorly and superiorly as it engages the thyroid cartilage. The scalpel can then be removed. Alternatively, a gum elastic bougie can be inserted into the trachea beside the incision to maintain the track. The bougie can also be preloaded with an endotracheal tube.

Use the Trousseau dilator, if available, to dilate the cricothyroid membrane in both the sagittal and transverse planes.

Select an endotracheal tube usually one size less than for normal intubation as well if available. Lubricate and insert perpendicular to the trachea, rotating the Shiley while inserting it.

Inflate the cuff and begin ventilating. Then, confirm appropriate placement and secure the endotracheal tube in place.

## 4. EAR LACERATION

Scott C. Sherman



**FIGURE 1.** Laceration of the pinna of the ear.

### Clinical Presentation

Result from blunt or sharp trauma

Auricular cartilage is avascular, whereas the overlying skin is extremely vascular.

### Diagnosis

Diagnosis is based solely on the physical examination.

### Management

Avoid debridement of soft tissue so that exposed cartilage can be completely

covered.

Consult a plastic surgeon for cartilage defects >5 mm, amputation, or inability to cover exposed cartilage.

Repair simple lacerations with interrupted 6-0 nonabsorbable suture. The suture should go through soft tissue and perichondrium.

Following repair, apply a dressing that provides support for the ear and prevents the development of an auricular hematoma.

Antibiotics are recommended to prevent the development of chondritis when cartilage has been exposed or a hematoma has been drained.

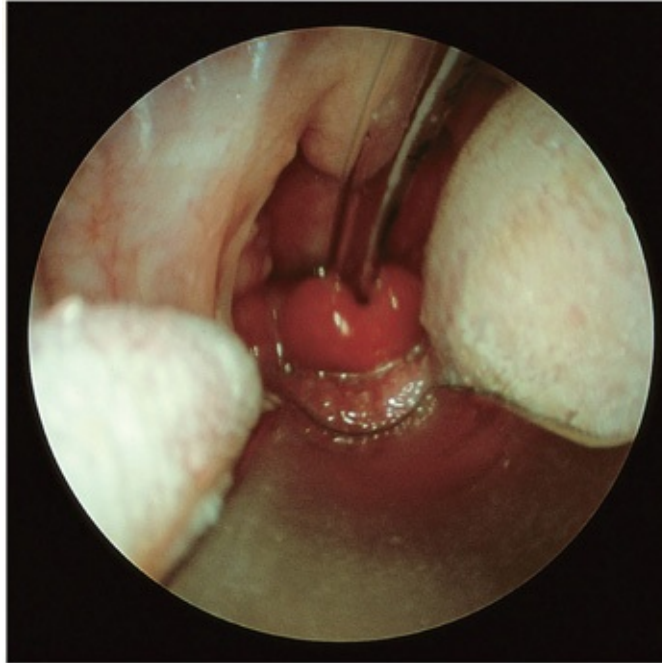
## 5. EPIGLOTTITIS

Basem F. Khishfe



**FIGURE 1.** Thumbprint sign on lateral radiograph of the neck (arrow).





**FIGURE 2.** Direct visualization of the inflamed, "cherry red" epiglottis. (From Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)

## Clinical Presentation

An acute illness characterized by severe inflammation and edema of the supraglottic structures, epiglottis, vallecula, arytenoepiglottic folds, and arytenoids that can rapidly progress to airway obstruction and potentially death

Incidence has decreased since the introduction of the *Haemophilus influenzae* type b vaccine.

Can present at any age; however, postvaccine era is more common in adults. Usually presents with an upper respiratory infection–like prodrome

## Diagnosis

Diagnosis may be confirmed with a lateral soft tissue neck x-ray showing the classic thumbprint-like epiglottitis.

In a cooperative patient not in moderate/severe distress, direct visualization using nasopharyngoscope may be performed.

Computed tomography should be reserved for patients with no airway distress and no evidence of epiglottitis on x-ray.

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## Management

Patients in severe respiratory distress should not be disturbed for the purpose of radiographic evaluation and attempts to visualize the epiglottis in the emergency department.

Allow patients to sit in their most comfortable position to breathe.

Set up airway equipment at bedside.

Immediate ENT and anesthesia consult

Intravenous hydration and antibiotics

Admit patient to intensive care unit setting or directly to the operating room to secure airway.



## 6. EPISTAXIS

Bryan Beaver, Jason P. Stopyra



FIGURE 1. Epistaxis from Kiesselbach's plexus.

### Clinical Presentation

It is estimated that 60% of the general population will report a nosebleed in their lifetime with less than 10% seeking medical attention.

The most common causes include upper respiratory infection, rhinosinusitis, and trauma. Recurrent epistaxis should raise concern for a neoplastic process. Anticoagulation with warfarin, aspirin, or other blood thinners is frequently associated with epistaxis.

Anterior epistaxis, most commonly involving Kiesselbach's plexus on the nasal septum, accounts for 90% of all nosebleeds. Posterior epistaxis comprises the remaining 10% and typically involves a branch of the sphenopalatine artery.

### Diagnosis

Initial assessment should include rapid evaluation for potential airway

compromise and hemodynamic instability.

Assemble all necessary materials including a light source, nasal speculum, suction catheter, packing materials, and vasoconstrictor/anesthetic medications prior to beginning examination.

Bleeding from both nares, inability to identify an anterior source, or continued posterior bleeding after an appropriately placed anterior nasal pack suggests posterior epistaxis.

Consider checking prothrombin time/international normalized ratio and complete blood count when indicated.

## Management

Have the patient clear clots by blowing the nose. Instill a topical vasoconstrictor such as oxymetazoline or phenylephrine, followed by application of bilateral pressure on the nasal septum for 10 to 15 minutes.

If direct pressure fails to achieve hemostasis and a bleeding site can be identified, chemical cauterization with silver nitrate may be attempted.

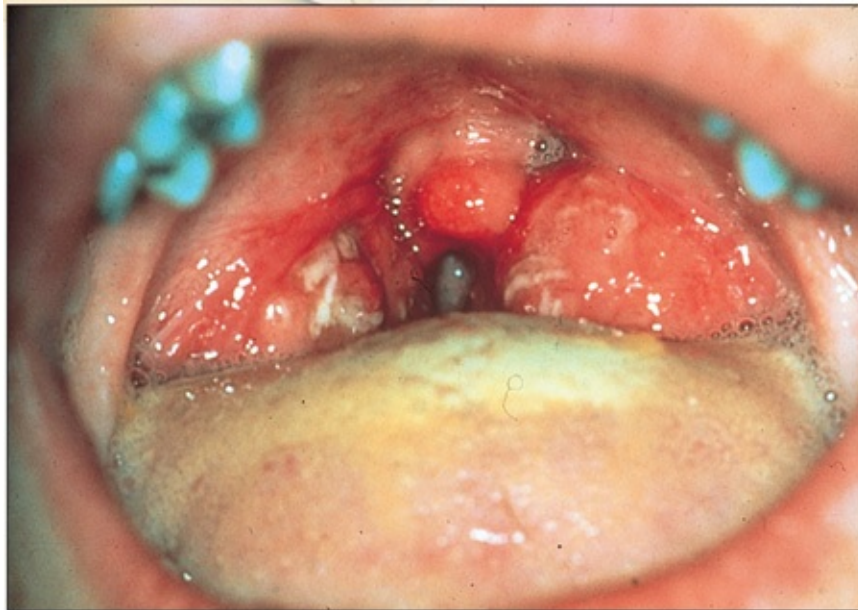
Cautery should not be attempted on both sides of the septum because this could lead to necrosis.

If bleeding persists, anterior nasal packing may be placed with preformed nasal tampon, nasal sponge, or epistaxis balloon. Follow-up should be arranged in 48 to 72 hours for packing removal. Antibiotic prophylaxis after packing continues to be recommended to prevent toxic shock syndrome and other complications.

For posterior epistaxis, use either a commercially designed posterior pack or 12 to 14F Foley catheter. Patients requiring posterior packing should have ENT consultation and admission to the hospital due to higher complication rates.

## 7. EXUDATIVE PHARYNGITIS

John Moorhead, Colby Austin



**FIGURE 1.** Exudative pharyngitis. (Photo courtesy of American Academy of Pediatrics.)

### Clinical Presentation

Fever, sore throat, and odynophagia are most common complaints. Associated symptoms of an upper respiratory infection may be present. Patients should not have any airway compromise, uvular deviation, or trismus; which would be more indicative of deeper tissue infections such as peritonsillar abscess, submandibular/retropharyngeal space infections, and epiglottitis.

### Diagnosis

Diagnosis is made by history and physical exam alone. Must be able to rule out life threats such as epiglottitis, peritonsillar abscess, submandibular space infections, and retropharyngeal space infections. Time course and risk factors are important elements of history.

## Management

Typically bacterial in etiology; rarely can be associated with viral etiologies such as acute infectious mononucleosis

Most common organism is group A  $\beta$ -hemolytic *Streptococcus* (GABHS). Penicillin is considered first-line treatment.

If GABHS is suspected, use Centor criteria to determine if treatment with antibiotics is warranted. Centor criteria include tonsillar exudates, tender anterior cervical lymphadenopathy, absence of cough, and fever. For patients with two or more criteria, consider rapid strep test. For patients with three or more criteria, consider empiric antibiotic treatment.

Be vigilant for infection due to atypical organisms such as gonococcal pharyngitis or diphtheria from *Corynebacterium diphtheria* in high-risk patients.

## 8. LIP LACERATION

Scott C. Sherman



**FIGURE 1.** Laceration through the vermilion border of the lip.



**FIGURE 2.** This laceration extended through to the oral mucosa.

### Clinical Presentation

May be due to blunt or sharp trauma; frequently due to a fist

Special attention should be made to determine whether the laceration involves the vermilion border or extends through the skin to the oral mucosa.

## Diagnosis

Diagnosis is based on a thorough physical examination.

Radiographs are only necessary if there is concern for a mandible fracture or that a tooth avulsed and entered into the soft tissues.

## Management

Anesthetize the lip using a mental or infraorbital nerve block. This avoids tissue distortion that occurs with local anesthetic into the wound.

Irrigate with saline.

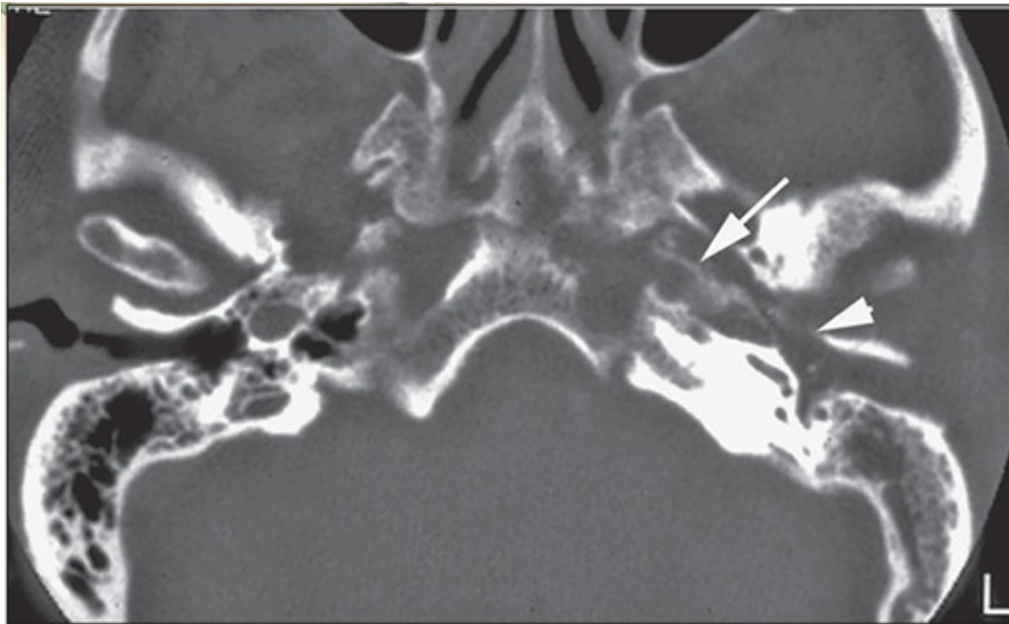
Start the repair by approximating the vermilion border with a single 6-0 nonabsorbable suture. Next, close the muscle layer using 5-0 absorbable suture. Then, close the mucosal layer with 5-0 absorbable suture. Finally, close the skin with interrupted 6-0 nonabsorbable suture.

Prophylactic antibiotics are controversial.



## 9. MALIGNANT OTITIS EXTERNA

Ashley Zielinski, Craig Warden



**FIGURE 1.** Malignant otitis externa. A diabetic patient with necrotizing otitis externa with CT showing erosion of the petrous (arrow) and tympanic (arrowhead) portions of the temporal bone. (From Mancuso AA, Hanafee WN. *Head and Neck Radiology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Patients with malignant otitis externa usually present with severe otalgia and otorrhea. The pain often extends into the temporomandibular joint and jaw. Physical examination reveals swelling and redness around the ear with granulation tissue often visible in the external auditory canal at the bone–cartilage junction.

Typically occurs in immunocompromised and diabetic patients

Most commonly caused by *Pseudomonas aeruginosa*

Cranial nerves VII to XII may be affected as the infection progresses.

### Diagnosis

Diagnosis is often missed due to the similarity to simple otitis externa in the

initial phases.

High resolution computed tomography (CT) of temporal bone shows bony erosion.

Magnetic resonance imaging (MRI) is more sensitive than CT for detection in the early phases.

## Management

Requires prolonged intravenous antibiotics focused on pseudomonal coverage followed by oral antibiotics; biopsy may be performed to guide antibiotic selection and rule out malignancy.

Microsuction of the area, topical antibiotics, and appropriate glycemic control are important components to successful treatment.

ENT consultation is recommended.

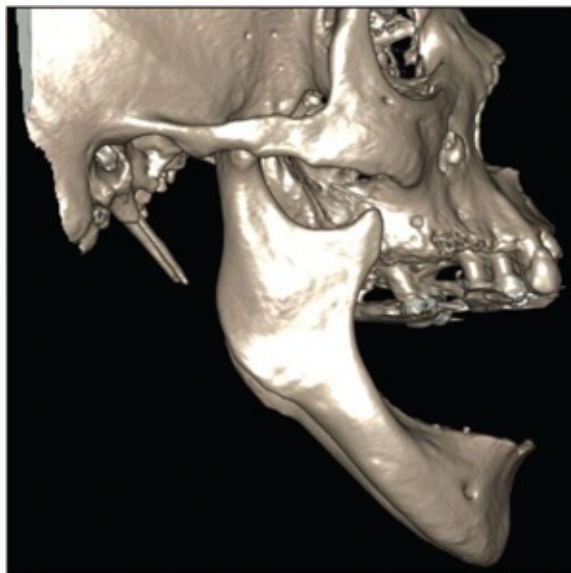


## 10. MANDIBULAR DISLOCATION

Michael Gottlieb



**FIGURE 1.** Open mouth in a patient with mandibular dislocation.



**FIGURE 2.** The condyle is dislocated anteriorly becoming locked in the anterior aspect of the eminence.

Clinical Presentation

Most commonly occurs after yawning, laughing, singing, dental extraction, and seizures

Usually bilateral mandibular dislocation is most common, but unilateral dislocation can occur with the jaw deviating to the opposite side.

Anterior dislocations are the most frequent and consist of anterior displacement of the mandibular condyle out of the temporomandibular fossa.

The condyle becomes locked in the anterior superior aspect of the eminence, resulting in spasm of the masseter, temporalis, and pterygoid muscles.

Posterior, superior, and lateral dislocations may also occur but are generally traumatic in etiology.

Patient will typically present with an open mouth, difficulty speaking, excessive salivation, and jaw malocclusion.

## Diagnosis

When this occurs spontaneously with a classic history, the diagnosis is clinical.

If the dislocation is traumatic, obtain a panoramic radiograph of the jaw (Panorex) or computed tomography scan to evaluate for concomitant fracture.

All patients with mandibular dislocations should have a thorough evaluation of their cranial nerves for associated injury.

## Management

The standard reduction technique is performed on a seated or prone patient, by placing gauze-wrapped thumbs over the occlusal surfaces of the patient's inferior molars and applying slow downward pressure, followed by backward pressure.

The wrist pivot method is performed while facing the patient, by placing the thumbs on the mentum and fingers along the occlusal surfaces of the patient's inferior molars and applying slow pivoting of the wrists with the thumbs moving in a cephalad direction.

Parenteral analgesics and massage of the spasmodic muscles may facilitate the reduction attempt.

Successfully reduced patients may be discharged with a soft diet and

precautions to avoid opening their mouth wider than 2 cm over the subsequent 2 weeks.

Subspecialty consultation should occur with patients that fail reduction techniques, multiple prior dislocations, or if there is an associated fracture with the dislocation.

## 11. NASAL SEPTAL HEMATOMA

Mary P. Chang



FIGURE 1. Bilateral nasal septal hematomas.

### Clinical Presentation

Can occur after trauma or surgery, commonly with nasal bone fractures  
Usually associated with nasal deformity, epistaxis, and occasionally no signs of trauma  
Can be unilateral or bilateral

### Diagnosis

Examine both nares with an otoscope or nasal speculum.  
Palpate the entire septum in case visualization misses the hematoma.  
Hematoma may be a bluish or flesh-colored fluctuance seen emanating from the medial septal wall.

### Management

Hematoma must be drained in the emergency department to preserve the nasal

septum. Failure to do so may lead to septal necrosis and saddle nose deformity.

Use topical or injectable lidocaine to anesthetize the hematoma.

Aspirate clot with a wide-bore needle or make a scalpel incision to evacuate the clot.

Pack anterior nares with Vaseline gauze or nasal tampon so that there is pressure to avoid hematoma formation and necrosis of the septum.

## 12. OTITIS EXTERNA

Ashley Zielinski, Craig Warden



FIGURE 1. Normal appearance of the ear.



**FIGURE 2.** Erythema and edema of the external auditory canal, otitis externa.

## Clinical Presentation

Inflammation of the external auditory canal leading to otalgia, varying degrees of hearing loss or tinnitus, ear fullness, itching, and ear discharge

Precipitated by ear trauma such as cotton swab use, hearing aid use, or water exposure leading to secondary infection (which may be bacterial, viral, or fungal)

## Diagnosis

Ear pain is made worse with palpation of the tragus or traction of the pinna. Canal appears inflamed and narrowed with exudate and drainage.

## Management

If there is severe swelling of the canal, placement of an ear wick is necessary to ensure that treatment is able to penetrate the ear. Irrigation and suction of the external auditory canal may also be necessary to clear out exudates.

Treat with a 1-week course of steroid and antibiotic ear drops. The best bacterial coverage in adults is with a fluoroquinolone.

If refractory to treatment, may need to send culture to direct antibiotic selection

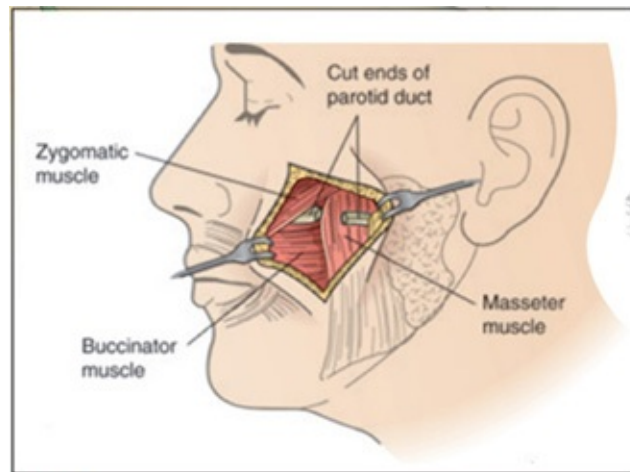
Fungal infections require 3- to 4-week courses of antifungal drops.

If the infection progresses to a cellulitis of the pinna and surrounding soft tissues, consider admission for intravenous antibiotics.



## 13. PAROTID DUCT LACERATION

Ashley Zielinski, Craig Warden



**FIGURE 1.** Anatomical depiction of a parotid duct laceration. (From Peitzman AB, Schwab CW, Yealy DM, et al. *The Trauma Manual: Trauma and Acute Care Surgery*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)



**FIGURE 2.** A line connecting the tragus and midportion of the upper lip approximates the location of the parotid (Stensen's) duct.

### Clinical Presentation

Any intraoral laceration should be evaluated for involvement of the parotid duct, salivary glands, and submandibular duct. The orifice of parotid duct is

located across from the second superior molar.

Typically associated with penetrating wounds of the cheek, especially if the wound falls lateral to a line vertical to the pupil and inferior to a line horizontal to the tragus

Blunt trauma may also tear the duct, resulting in a salivary pseudocyst that causes local swelling.

Suspect if saliva is present in the wound or if there is weakness of the upper lip with attempted puckering.

## Diagnosis

The duct is typically visualized through the wound superior to a small branch of the facial nerve and just inferior to a small accompanying artery.

If there is diagnostic uncertainty, expert consultation is warranted. The orifice of the duct can be cannulated with a lacrimal probe or small catheter.

Sialography is also sometimes used.

Beware of additional nearby injuries including lacerations to the muscles of mastication or facial expression as well as the underlying bony structures; advanced imaging may be necessary.

## Management

Most minor ductal lacerations heal without complications other than temporary saliva leakage.

Lacerations involving the parotid duct should be immediately repaired to avoid formation of a salivary fistula or sialocele.

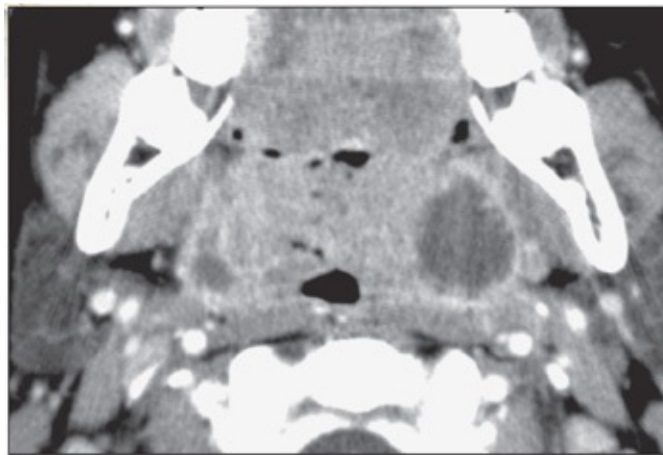
Some parotid duct injuries can have a delayed repair by a few days on the advice of and subsequent follow-up by ear, nose, and throat or facial plastic surgery specialist.

## 14. PERITONSILLAR ABSCESS

John Moorhead, Colby Austin



**FIGURE 1.** Note the bulging of the peritonsillar tissues. (Courtesy of Seth Zwillenberg.)



**FIGURE 2.** Peritonsillar abscess CT images. (From Mancuso AA, Hanafee WN. *Head and Neck Radiology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Fever, severe increasing, often unilateral sore throat, odynophagia, and malaise are most common complaints.

Adjacent tonsil will be inferiorly and medially displaced.

Trismus and contralateral deflection of uvula are typically seen on physical

exam.

Assess for airway difficulties, although rare, and ability to handle secretions.

## Diagnosis

Diagnosis is often made by history and physical examination alone.

When diagnosis is in question, needle aspiration of purulent material, bedside ultrasound using intracavitary probe, or contrast-enhanced computed tomography (CT) may help confirm diagnosis.

Contrast-enhanced CT is indicated if there is concern for spread of infection beyond the peritonsillar space.

## Management

Treatment involves either needle aspiration or incision and drainage.

Procedural ultrasound guidance may be used for identification of the abscess and surrounding vascular structures.

Anesthetize with lidocaine or benzocaine-tetracaine spray followed by 1 to 2 mL of lidocaine directly injected into the mucosa of anterior tonsillar pillar.

Aspiration needle should penetrate no more than 1 cm to avoid internal carotid artery.

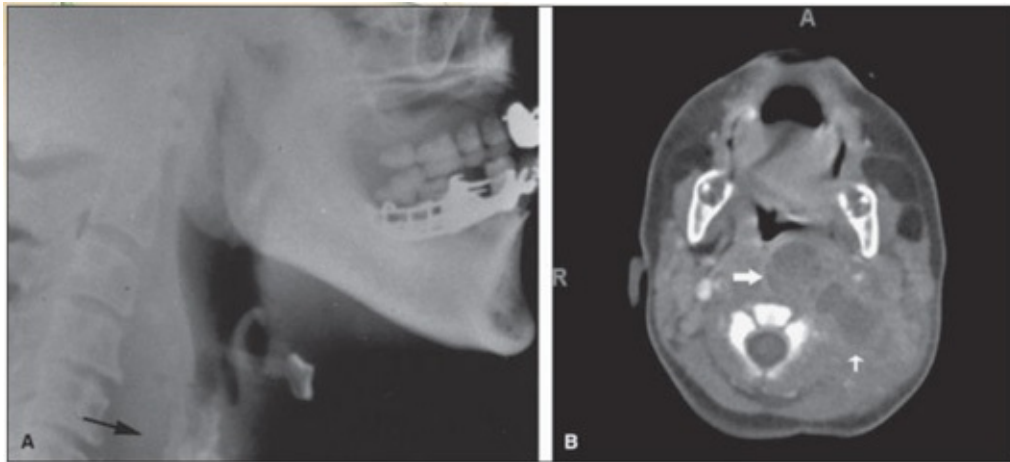
Internal carotid artery usually lies laterally and posterior to the posterior edge of the tonsil.

Typically polymicrobial infections, with group A *Streptococcus* as the most commonly identified organism

Provide 24-hour follow-up or observation for airway complications and intravenous antibiotics and pain management.

## 15. RETROPHARYNGEAL ABSCESS

John Moorhead, Colby Austin



**FIGURE 1.** Retropharyngeal abscess, both CT and plain film (arrows). (From Fleisher GR, Ludwig S. Textbook of Pediatric Emergency Medicine. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Fever, sore throat, and dysphagia are most common complaints, which are often difficult to distinguish from uncomplicated pharyngitis.

As the disease progresses, symptoms of airway inflammations and obstruction develop with symptoms such as neck pain, difficulty swallowing, torticollis and inability to extend the neck, muffled voice, trismus, chest pain, and rarely respiratory distress.

Stridor and neck edema are typically seen in children but not in adults.

### Diagnosis

In children, lateral soft tissue radiograph of the neck can demonstrate thickening and protrusion of the retropharyngeal wall.

Contrast-enhanced computed tomography (CT) scan of the neck is the preferred imaging study in adults.

Early CT findings may reflect reactive nonsuppurative edema, mild fat stranding with discernible tissue planes, and no associated enhancement.

Do not send a patient with airway distress for unobserved CT scanning.

## Management

All patients need immediate consultation with an otolaryngologist to prevent an airway catastrophe.

Any patients with airway compromise should go immediately to the operating room for drainage.

Antibiotic treatment should be started in the emergency department; typically polymicrobial infections. Regimens include clindamycin or cefoxitin; alternatively, may also use Zosyn or Unasyn.

A few patients with small abscesses can be managed with intravenous antibiotics alone; however, majority will require surgical intervention.

Complications include extension of the infection into the mediastinum and upper airway asphyxia from direct pressure or aspiration after sudden rupture of abscess.



## 16. SIALOLITHIASIS

Emily Carlisle



**FIGURE 1.** Patient with sialolithiasis involving the right parotid gland.

### Clinical Presentation

Saliva from the submandibular gland emerges near the frenulum via Wharton's duct; from the parotid glands, it emerges opposite second molar via Stensen's duct.

Upward of 80% of stones are found in the submandibular system, with the majority in Wharton's duct; of the stones arising from the parotid gland, about half are in the gland itself rather than Stensen's duct. The sublingual and minor salivary glands empty into Wharton's duct or directly into the mouth and rarely develop stones.

Risk factors: male gender, age 30 to 60 years, dehydration, diuretics or anticholinergics, smoking, gout, and trauma

Patients typically present with painful swelling worse during eating or anticipation of eating but may have only pain or swelling.

Palpation along the floor of the mouth while compressing the submandibular

gland or along buccal mucosa while compressing the parotid gland may reveal stone, tenderness of the gland, or decreased salivary output compared to contralateral side. Purulent drainage or erythema indicates likely bacterial sialadenitis.

## Diagnosis

Palpation of hard, small stone on bimanual exam is highly suggestive if not confirmatory.

Consider noncontrast computed tomography or ultrasound if no stone palpated or other diagnoses must be ruled out.

Differential diagnosis: neoplasm, viral sialadenitis (HIV, mumps), autoimmune disease, malnutrition

Complications: abscess, chronic obstruction with decreased production and destruction of gland, Ludwig's angina (rare)

## Management

Initially conservative: hydration, warm compresses, gland massage, "milking" of duct, sialogogues (e.g., hard sour candy), and nonsteroidal antiinflammatory drugs

Consider cessation of anticholinergic medications (e.g., diphenhydramine, amitriptyline).

Oral antistaphylococcal coverage (e.g., cephalexin or dicloxacillin 500 mg four times daily) if concern for concomitant bacterial sialadenitis and patient is nontoxic and otherwise appropriate for discharge



## 17. THRUSH

Michael Gottlieb



**FIGURE 1.** Oral thrush in an infant. (Courtesy of Paul S. Matz, MD.)



**FIGURE 2.** Oral thrush in an adult.

## Clinical Presentation

Thrush is typically limited to infants and neonates, patients on antibiotics or steroids, and patients with immunodeficiencies.

Thrush occurs when the normal host immunity or host flora are disrupted, allowing for overgrowth of *Candida albicans*.

Lesions often begin as small, focal areas and enlarge to white patches on the oral mucosa.

## Diagnosis

Thrush is a clinical diagnosis.

The diagnosis can be confirmed with a Gram stain or potassium hydroxide preparations of a scraping.

Differentiate thrush from other lesions by scraping it with a tongue blade.

Thrush will be difficult to remove and will leave behind a tender, inflamed base.

Plaques can be cultured, but this is rarely indicated.

## Management

Treatments include the following:

Nystatin oral suspension 400,000 to 600,000 units, swish and swallow, four times daily until 48 hours after symptoms resolve

Clotrimazole 10 mg troches, five times daily for 2 weeks

Fluconazole 200 mg pill on day 1, followed by 100 mg daily for 2 weeks

Thrush is typically self-limited, and patients may be discharged home unless concomitant illness requires further evaluation.

## 18. THYROGLOSSAL DUCT CYST

Devin Keefe



**FIGURE 1.** Patient with a thyroglossal duct cyst. Note the midline location.

### Clinical Presentation

Thyroglossal duct cysts (TGDCs) are the most common congenital neck mass, resulting from a swelling of the thyroglossal duct, a tract of epithelium connecting the foramen cecum to the pyramidal lobe of the thyroid. The duct, a remnant of the embryologic migration of the thyroid, persists due to a failure to atrophy.

TGDCs are fluctuant, round, and painless masses of the midline, anterior neck that may present at any age; most are infrahyoid or at the level of the hyoid. Movement of the mass is synchronous with swallowing or tongue protrusion. TGDCs do not transilluminate.

Pain or tenderness on exam indicates infection or hemorrhage.

## Diagnosis

On ultrasound, cysts are anechoic and homogenous if uninfected.

Heterogeneity of cyst contents suggests infection.

Computed tomography offers better visualization than magnetic resonance imaging for surgical planning.

Consider thyroid function tests to uncover occult hypothyroidism, which may indicate that the cyst contains ectopic thyroid tissue.

A normal thyroid must be confirmed on imaging. If presurgical imaging is abnormal, thyroid-stimulating hormone is elevated, or there is clinical evidence of hypothyroidism (constipation, weight gain, somnolence, growth delay, etc.), then a radionuclide thyroid scan should be performed before surgery to rule out ectopic thyroid or accessory thyroid nodule.

## Management

Surgical removal is the definitive treatment.

Antibiotics are used to treat infected cysts prior to excision.

Thyroid replacement therapy may be necessary postoperatively if glandular tissue is excised.

## 19. TYMPANIC MEMBRANE RUPTURE

Christopher J. Tegeler, Jason P. Stopyra



**FIGURE 1.** TM rupture. (Courtesy of Dan Kirse, MD, FAAP, Wake Forest University School of Medicine, Winston-Salem, NC.)

### Clinical Presentation

Patients present acutely with mild to severe ear pain, hearing loss, tinnitus, vertigo, and/or bloody/purulent otorrhea.

#### Etiology

Direct trauma—accidental perforation from instrumentation such as cotton-tipped swab, pen, or other foreign body

Barotrauma—ambient pressure changes from flying or diving; acute pressure changes seen in explosions and open palm slap to the side of the head

Infection—acute otitis media most commonly; rarely acute necrotic myringitis

### Diagnosis

Carefully clean out debris or blood from external ear canal with instrument or suction. *Do not* irrigate because it may push fluid and debris into the middle ear.

Completely visualize the tympanic membrane (TM) with an otoscope.

Insufflation showing an immobile TM may be required to detect a small rupture.

If head trauma is likely the etiology, obtain imaging to rule out temporal bone fracture and assess for intracranial injury.

Test gross hearing and then use Weber/Rinne test to delineate conductive versus sensorineural.

## Management

Instruct patient not to get ear canal wet—can use a petroleum jelly-soaked cotton ball to prevent water from entering the external ear canal during bathing.

TMs usually heal in several days to a month with conservative management and infection treatment/prevention in 80% to 90% of cases.

Oral antibiotics commonly used for the treatment of otitis media are recommended.

Otological fluoroquinolones may also be used.

Analgesics

Urgently consult an otolaryngologist, if vertigo, sensorineural hearing loss, severe conductive hearing loss, active bleeding, debris that cannot be removed, or facial nerve paralysis are present.





SECTION

E

DENTAL

SECTION EDITOR  
Erik Nordquist



# 1. ACUTE NECROTIZING ULCERATIVE GINGIVITIS

Joseph D. Grubic, Michael E. Nelson



**FIGURE 1.** Ulceration and gingival edema. (From Lippincott Williams & Wilkins. *Comprehensive Dental Assisting*. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

## Clinical Presentation

Typically occurs as complication of chronic gingivitis from the soft tissue invasion of multiple organisms  
Characteristics include foul breath, blunting of the gingival papilla between teeth, and necrosis of the gingiva.  
Classically affects young individuals with poor oral hygiene  
Smoking, immunosuppression, and malnutrition are risk factors.  
Look for signs signifying systemic infection, such as fever and lymphadenopathy.

## Diagnosis

Severe, acute onset of pain is the hallmark of the disease.  
Ulceration of interdental papillae with marked gingival edema is typically present.

Debridement of the necrotic material will cause bleeding and expose erythematous and ulcerative gingiva.

## Management

Referral to a dentist or periodontist for definitive treatment and debridement under anesthesia

Immediate emergency department treatment includes antimicrobial rinses, such as chlorhexidine, together with penicillin or metronidazole for 7 to 10 days.

Additional antimicrobial options include clindamycin or doxycycline.

Provide topical anesthetic (i.e., lidocaine) and oral analgesics so that patient may initiate proper oral hygiene (brushing, flossing).

## 2. DENTAL ABSCESS AND INFECTION

Oscar Bravo



FIGURE 1. Buccal space dental abscess.

### Clinical Presentation

Suppurative dental infections may spread into potential fascial planes in the orofacial area.

Orofacial infections can involve the buccal, submental, masticator, canine, and infratemporal spaces.

The location of the infected space can help identify the underlying infected tooth.

There is potential for contiguous spread to deeper fascial planes of the face and neck.

### Diagnosis

Facial swelling or asymmetry is usually present.

Trismus can suggest a masticator space infection.

Computed tomography imaging is sensitive for osseous structures and can be

helpful for evaluation of deep space infections.

## Management

Assessment and management of the airway as clinically indicated

Surgical drainage is the primary treatment modality.

Oral surgery consultation is indicated for complicated odontogenic infections.

Antibiotic therapy with activity against oral flora should be considered, particularly in patients with signs of systemic involvement or with immunocompromised states.

### 3. DENTAL AVULSIONS

Elena Strunk, Michelle Sergel



FIGURE 1. Tooth avulsion.



FIGURE 2. Tooth reimplantation.

#### Clinical Presentation

Tooth avulsion is the total displacement of a tooth from its socket due to tearing of the periodontal ligament.

The patient presents with a tooth separated from the socket.

Significant force is required to avulse a tooth. Other dental fractures are common.

## Diagnosis

Tooth avulsion is diagnosed clinically and radiographically.

The clinical examination should include examination of the tooth socket to determine if it is intact and if the tooth can be reimplanted.

Primary teeth are typically not reimplanted.

## Management

Rinse the tooth with sterile saline.

Handle only the crown of the tooth. Survival of the periodontal ligament fibers that remain attached to the root of the avulsed tooth is very important to ensure successful reimplantation.

Remove the clot from the tooth socket and irrigate with normal saline.

Anesthetize the area locally if clinically indicated.

Reimplant the tooth with firm pressure and have the patient bite on gauze.

Consider the application of periodontal dressing.

If unable to reimplant the tooth, place it in storage medium (Hank's balanced salt solution or sterile saline) until the tooth can be reimplanted.

Refer the patient to a general dentist or oral surgeon for permanent stabilization as soon as possible.

Patients should receive prophylactic antibiotics and antimicrobial rinses for 7 to 10 days.



## 4. DENTAL CARIES

Joseph D. Grubic , Michael E. Nelson



FIGURE 1. Severe dental caries of premolar.



FIGURE 2. Dental caries of the incisors.

### Clinical Presentation

Can be seen as a pits, fissures, or discolorations on the surface of teeth  
Represents a major cause of tooth loss in individuals <35 years old

### Diagnosis

May be asymptomatic or the affected tooth may be sensitive to hot/cold foods

and percussion

Sensitivity to percussion of the tooth likely represents progression to pulpitis or periapical abscess.

## Management

Prevention is key and can be accomplished with regular brushing (two to three times a day) and avoidance of sugar-rich foods and beverages.

If a periapical abscess is suspected, the patient can be discharged home on penicillin or clindamycin (if penicillin allergic) with chlorhexidine mouth wash.

Refer to a dentist for definitive treatment.



## 5. DENTAL FRACTURE

Ellica Chu, Deborah Kimball



**FIGURE 1.** Dental fracture of pediatric tooth. (From Lippincott Williams & Wilkins. *Comprehensive Dental Assisting*. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.)



**FIGURE 2.** Dental fracture of adult tooth. (Photograph courtesy of Dr. Stephen Rosenstiel, Section Head of Restorative and Prosthetic Dentistry, Ohio State University.)

### Clinical Presentation

Patients will present with a chipped or broken tooth after a fall or facial trauma.

Seventy percent of dental fractures involve the dentin.

### Diagnosis

Ellis classification

Ellis class I fractures (enamel fractures)—includes chipping, cracks, or

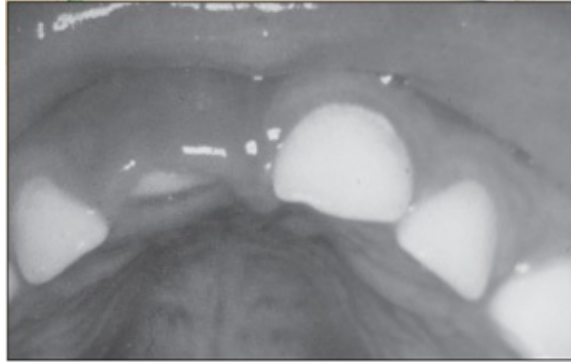
incomplete fractures through the enamel. These fractures are not painful. Ellis class II fractures (enamel and dentin fractures)—most common type. The deep yellow color of dentin can be seen. Patients will be sensitive to percussion, forced air, and temperature differences. Ellis class III fractures (enamel, dentin, and pulp fractures)—exposed dentin will have a pink blush or there will be blood from the pulp cavity. These fractures may not be painful initially if the nerve is concussed. Ellis class IV fractures (root fractures)—coronal segment of the tooth may be mobile, and tooth is usually tender to percussion; can confirm fracture with dental radiographs.

## Management

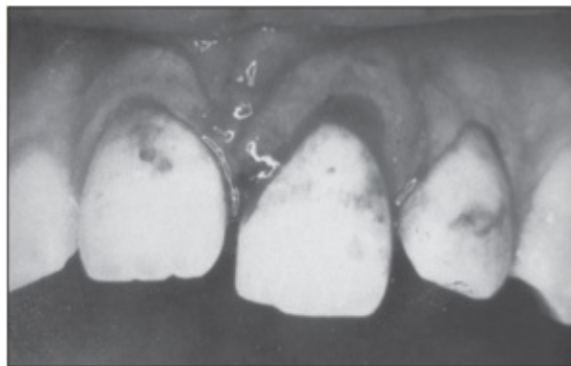
Treatment is aimed at sealing dentin tubules and to prevent pulp necrosis. Ellis class I fractures—smooth sharp corners and refer to a dentist for aesthetic repair in 1 to 2 weeks. There is a 3% to 5% chance of pulp necrosis. Ellis class II fractures—block the tooth, dry the fracture, and cover the exposed dentin with calcium hydroxide paste or dental sealer. Patient should follow up with a dentist within 24 hours. There is a 7% to 10% chance of pulp necrosis if not treated within 1 week. Ellis class III fractures—block the tooth, dry the fracture, and cover the fracture with calcium hydroxide paste or dental sealer. Pulp necrosis is likely, and patients require follow-up with a dentist within 24 hours; will require a root canal. Ellis class IV fractures—reposition the coronal segment to its original position, which can be confirmed using radiographs, and stabilize the tooth with periodontal paste or orthodontic bands with calcium hydroxide paste along the fracture line. Follow up with a dentist in 1 day. Consider the presence of associated facial fractures or aspiration of tooth fragments.

## 6. DENTAL LUXATIONS

Elena Strunk, Michelle Sergel



**FIGURE 1.** Intrusive luxation of a central incisor. (From Wolfson AB, Cloutier RL, Hendey GW, et al. *Harwood-Nuss' Clinical Practice of Emergency Medicine*. 6th ed. Philadelphia, PA: Wolters Kluwer Health; 2015.)



**FIGURE 2.** Extrusive luxation of a central incisor. (From Wolfson AB, Cloutier RL, Hendey GW, et al. *Harwood-Nuss' Clinical Practice of Emergency Medicine*. 6th ed. Philadelphia, PA: Wolters Kluwer Health; 2015.)

### Clinical Presentation

There are five types of luxations: concussion, subluxation, extrusive luxation, lateral luxation, and intrusive luxation.

A tooth with a concussion is tender to percussion but does not demonstrate any loss of mobility.

A subluxed tooth has increased mobility without evidence of dislodgment on imaging.

An extrusive luxation presents with a tooth appearing elongated, tender, and excessively mobile. It is a tooth that is partially avulsed from the alveolar bone.

A lateral luxation presents with the displacement of the tooth in any direction

other than axial. Displacement is accompanied by comminution or fracture of alveolar bone.

An intrusive luxation is the displacement of the tooth into the alveolar bone. This is accompanied by comminution or fracture of the alveolar socket.

## Diagnosis

Diagnosis is made primarily clinically.

Radiographs may aid in the diagnosis and be of particular interest to dental consultants.

## Management

Management depends on the tooth involved, severity of injury, and presence of other fractures.

Tooth concussion and subluxation is treated with nonsteroidal antiinflammatory drugs (NSAIDs), soft diet, and referral to a general dentist. Extrusive luxations require repositioning of the tooth to its original position with axial digital pressure and splinting to stabilize the tooth.

A lateral luxation can be challenging to reposition. One must manipulate the displaced tooth with thumb and forefinger. If the alveolar fracture is minimal, temporary splinting with a periodontal dressing is acceptable. Otherwise, an oral surgeon or dentist needs to splint the injury urgently.

Intrusive luxations are more serious due to the more significant damage to the alveolar socket and periodontal ligament. Management is determined by a dental specialist.

All patients with extrusive, lateral, and intrusive luxations should follow up with a dentist or oral surgeon within 24 hours.

Advise patient to maintain a soft diet for 1 to 2 weeks and to use a soft brush and antimicrobial rinse.

## 7. LUDWIG'S ANGINA

Thomas Adam Criswell, Rashid Kysia



FIGURE 1. Significant edema under the mandible with tongue elevation in a patient with Ludwig's angina who required nasotracheal intubation.

### Clinical Presentation

Bilateral cellulitis involving submandibular, submaxillary, and sublingual spaces

The majority of cases are of odontogenic origin (second and third mandibular molars); a minority are from oral lacerations or piercings, mandibular fractures, and peritonsillar/parapharyngeal abscesses.

Children may present without obvious underlying cause.

Most patients have no comorbid disease, but the immunocompromised are at higher risk.

Hallmark signs are neck swelling with “woody” induration and tongue elevation/protrusion. Commonly also see fever, tooth pain, dyspnea, dysphagia, and trismus.

Signs of airway compromise can be subtle. Stridor, inability to swallow secretions, and cyanosis are late signs.

### Diagnosis

Diagnosis is based on history and physical exam.

Computed tomography can confirm diagnosis, evaluate for mass effect on airway, and plan potential surgical intervention in a stable patient with a

secured airway.

## Management

Airway compromise is primary concern. Otolaryngology, oral surgery, and anesthesia should be consulted immediately if available.

Patient should be maintained in sitting position with oxygen supplementation.

Airway observation is only recommended in select population with less severe disease and with no airway symptoms.

Due to difficulty of orotracheal intubation, fiberoptic nasotracheal intubation while awake is recommended with preparation for cricothyrotomy if needed.

Consider immediate transport to operating theater for surgical tracheostomy if available and patient stable.

Broad-spectrum antibiotics to cover aerobic and anaerobic bacteria should be implemented.

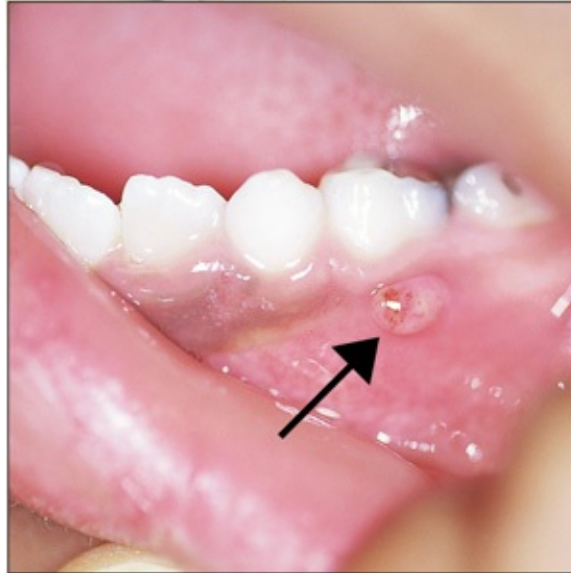
Dexamethasone intravenous may reduce edema and provide chemical decompression of the airway.

Admit to a critical care unit.



## 8. PULPITIS AND PERIAPICAL ABSCESS

Oscar Bravo



**FIGURE 1.** Localized gingival swelling (arrow) due to periapical abscess. (From Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)

### Clinical Presentation

Early symptom is usually acute, severe toothache that can be elicited by thermal changes, such as cold liquids or air.

Predisposing factors include dental caries or dental fractures, which can expose dentin to bacteria.

The involved tooth is often sensitive to percussion or palpation.

Buildup of necrotic tissue and debris at the apical foramen may lead to a periapical abscess.

### Diagnosis

Palpation or percussion tenderness of affected tooth

In the case of periapical abscess, gingival fluctuance or a draining sinus tract in the area of the root apex may be present.

Panoramic or periapical radiographs may reveal abscess; computed tomography imaging is usually not indicated.

## Management

Oral analgesics for pain

Although antibiotics are often prescribed, studies have not demonstrated improved outcomes in patients with pulpitis who receive antibiotics.

Antibiotics covering oral flora should be given to patients with a periapical abscess.

Referral to a dentist or oral surgeon for definitive treatment, such as root canal or extraction and drainage





SECTION

F

CARDIOVASCULAR

SECTION EDITOR

Christopher Ross

# 1. ABDOMINAL AORTIC ANEURYSM

Casey Wilson, Tiffany Fong

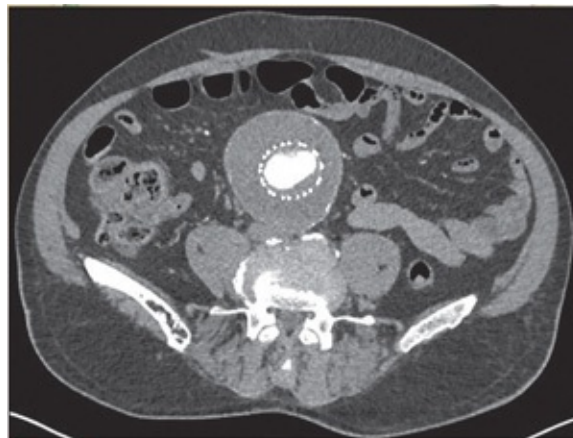


FIGURE 1. Abdominal aortic aneurysm CT: graft in place.



FIGURE 2. Abdominal aortic aneurysm: graft in place, transverse view.

## Clinical Presentation

Ruptured abdominal aortic aneurysm (AAA) is a leading cause of sudden death.

Symptoms include abdominal, flank, or back pain; limb ischemia; or constitutional symptoms such as fever or malaise. Less than 50% of AAA ruptures present with the triad of acute pain, hypotension, and a pulsatile abdominal mass.

On abdominal exam, the patient may have a widened aortic pulsation or

“pulsatile mass.” Palpation detects only 39% of AAAs. When rupture occurs, the mortality rate is 80%.

## Diagnosis

AAA is defined as full-thickness dilation of the aorta >50% above its normal diameter. This is variable depending on age, gender, and habitus but typically diagnosed at diameter >3.0 cm.

Ultrasound is the imaging modality of choice for an unstable patient with suspected AAA rupture. In experienced operators, ultrasonography has a sensitivity and specificity of nearly 100%. Patients with hypotension and ultrasonographic evidence of AAA should go directly to the operating room. Hemodynamically stable patients with suspected AAA should undergo computed tomography (CT) angiography or magnetic resonance imaging/magnetic resonance angiography. Findings of rupture include retroperitoneal hematoma and contrast extravasation.

## Management

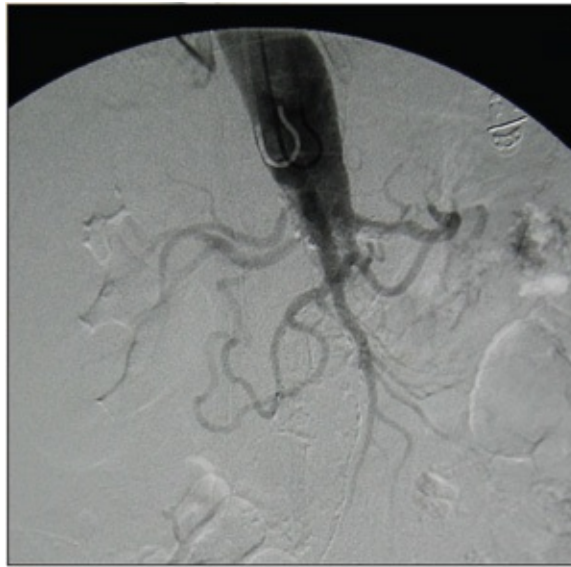
For a patient with abdominal pain and shock, bedside ultrasonography to evaluate AAA should be performed while resuscitation procedures are underway. Ruptured AAA requires emergent surgical consultation.

Hemodynamically stable patients with symptomatic AAA or signs of impending rupture should be hospitalized and observed.

In asymptomatic patients, elective AAA repair is indicated when the AAA exceeds 5.5 cm in diameter; this corresponds to increased risk of rupture.

## 2. ACUTE ARTERIAL OCCLUSION

Nathan Woltman, Sharon Bord



**FIGURE 1.** Acute aortic occlusion on angiography, Leriche's syndrome.

### Clinical Presentation

Acute occlusion most frequently occurs in the lower leg, often in patients with preexisting peripheral artery disease.

Acute occlusion causes limb ischemia and heralds tissue death. It is characterized by sudden severe pain, change in limb color and temperature, paresthesias, and loss of distal pulses.

Acute occlusion can represent sudden embolic occlusion of a healthy vessel, thrombosis of an atherosclerotic vessel, or external compression of a critically stenotic vessel.

Patients will often have coexisting risk factors for thrombosis or embolism, such as atrial fibrillation, a history of claudication, hypertension, diabetes, smoking, chronic inflammatory diseases, dyslipidemia, or hypercoagulable states.

## Diagnosis

Physical exam findings include a cool extremity, severe pain in affected limb, changes in skin color, and absent pulses.

If pulses are not palpable, a Doppler device should be used to verify.

If pulses are present by Doppler, an ankle-brachial index should be obtained.

Computed tomography angiography of the affected vessel will show occlusion, stenosis, or aneurysm.

Duplex ultrasound can be used to document vascular waveform and flow.

An electrocardiogram should be obtained to assess for atrial fibrillation.

## Management

The diagnosis of acute arterial occlusion requires emergent consultation with a vascular surgeon.

The patient should be anticoagulated with heparin.

Surgical or endovascular revascularization gives the greatest chance of limb salvage.

Bypass surgery, thrombolysis, angioplasty, endarterectomy, and stent placement are common treatment options. Amputation is the treatment of last resort.

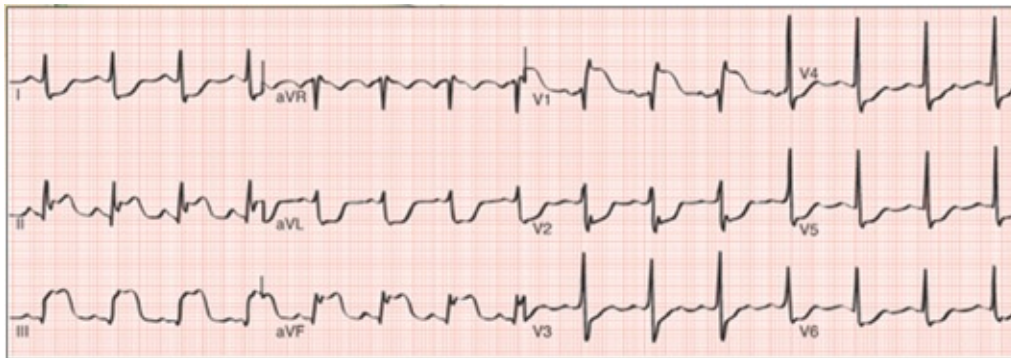
Choice of technique depends on presumed etiology of occlusion and patient comorbidities.

Adequate pain control should be provided.



### 3. ACUTE MYOCARDIAL INFARCTION

Nathan Woltman, Sharon Bord



**FIGURE 1.** Acute right ventricular myocardial infarction. ST-segment elevation is present in leads II, III, aVF, and V1; reciprocal ST-segment depression in all other leads. Note the discordant ST-segment elevation in V1 and depression in V2. (From Woods SL, Froelicher ES, Motzer SA, et al. *Cardiac Nursing*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.)

#### Clinical Presentation

The most common presentation involves chest pain, described as crushing or pressure, but atypical presentations are common especially in women, diabetics, and elderly.

Common associated symptoms include nausea, pain radiating to the arms or jaw, and diaphoresis.

Risk factors to consider include age, known coronary disease, diabetes, hypertension, hyperlipidemia, smoking, cerebral or peripheral vascular disease, family history of heart disease, recent cocaine use, and sedentary lifestyle.

Cause is an imbalance between myocardial blood flow and myocardial oxygen demand, either from a dynamic plaque rupture or a flow-limiting stenosis.

#### Diagnosis

ST-segment elevation myocardial infarction (STEMI) is defined as elevation at the J point of 2 mm in two contiguous precordial leads or 1 mm in contiguous limb leads.

Non-STEMI is characterized by either a normal electrocardiogram (ECG) or

nonspecific ECG changes such as T-wave inversions or ST-segment depression with positive cardiac biomarkers.

The syndrome of unstable angina refers to chest pain that is previously determined to be cardiac in nature occurring with increasing frequency or chest pain at rest with negative cardiac enzymes and no ST-segment elevation on ECG.

Although history and physical exam are important, ECG findings alone can make the diagnosis of acute STEMI.

Diagnosis is made by ECG and measurement of serial troponin levels.

## Management

The antiplatelet agent aspirin remains the cornerstone of treatment for acute coronary syndromes. Clopidogrel and GPIIb/IIIa inhibitors are increasingly being used.

Patients with strong suspicion for acute coronary syndrome, ST-segment elevation, or positive cardiac biomarkers should be anticoagulated with heparin or low-molecular-weight heparin.

Patients with ST-segment elevation on ECG should be transferred immediately to a cardiac catheterization lab to undergo percutaneous coronary intervention. If one is not available within 120 minutes, administer a thrombolytic agent.

## 4. AORTIC DISSECTION

Nathan Woltman, Rodney Omron



FIGURE 1. Ascending and descending aortic dissection (arrows).

### Clinical Presentation

Acute dissection of the aorta occurs when the vessel lumen separates, allowing blood to move from the lumen into the intima or media.

Dissection can be spontaneous or a result of trauma.

The classic presentation is a hypertensive patient with sudden onset of sharp or tearing pain in his or her chest and back, although sudden epigastric and back pain is a common presentation.

Possible exam findings include a peripheral pulse deficit, a “blowing” aortic murmur, hypertension, and neurologic abnormalities. Hypotension in the setting of aortic dissection is an ominous sign.

Risk factors include hypertension, age, atherosclerosis, Marfan syndrome, bicuspid aortic valve, aortic coarctation, previous heart or great vessel surgery, and disseminated syphilis.

### Diagnosis

Plain chest radiography is often obtained in patients with chest pain; a widened mediastinum suggests aortic dissection. Absence of mediastinal



abnormalities does not rule out aortic dissection.

Contrast-enhanced computed tomography scan of the aorta is diagnostic. If the patient has a contrast allergy, magnetic resonance angiography has similar sensitivity.

Aortography, transthoracic, and transesophageal ultrasound can be used in diagnosing aortic dissection. Transthoracic echocardiography has a low sensitivity for aortic dissection.

Electrocardiogram is nondiagnostic, although occasionally shows evidence of cardiac ischemia or ST-segment elevation if dissections extends through the origin of the coronary vessels.

## Management

Dissections are classified according to their location: Stanford A dissections involve the ascending aorta, and Stanford B dissections involve only the descending aorta.

Patients should have large-bore vascular access and be type and crossed for multiple units of blood.

Coagulopathies should be reversed, and platelets can be considered if the patient is on antiplatelet agents.

Both types of dissection are treated medically with a combination of blood pressure-lowering agents such as sodium nitroprusside and either  $\beta$ -blockers or calcium channel blockers to decrease sheer forces on the vessel wall.

Stanford A dissections require emergent surgical intervention, both open and endovascular techniques are commonly employed. Stanford B dissections can be managed surgically if medical intervention fails.

## 5. ATRIAL FIBRILLATION

Sharon Bord, Hardin Pantle

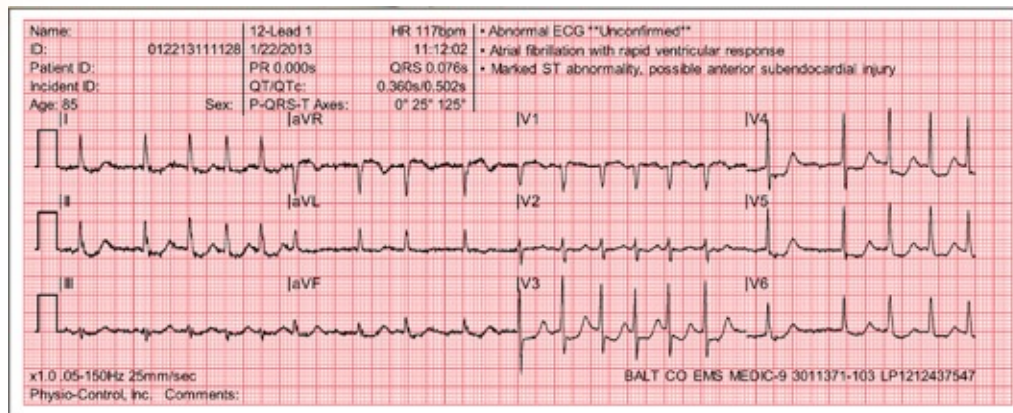


FIGURE 1. EKG demonstrating atrial fibrillation.

### Clinical Presentation

Common presentations include palpitations, light-headedness, shortness of breath, reduced exercise tolerance, presyncope, and chest pain.

Determining the timing of symptom onset may affect treatment. Patients sometimes remember exactly when they began to feel symptoms or palpitations; others might be in atrial fibrillation for an extended period of time and be asymptomatic.

The heart rate associated with atrial fibrillation can range from slow to very rapid. Atrial fibrillation with a rapid ventricular response (RVR) often has a heart rate  $>120$  bpm.

Some patients may experience hypotension due to poor filling and diminished forward flow of blood from the heart.

### Diagnosis

Electrocardiogram (ECG) will reveal absent P waves, irregularly irregular QRS complexes, and fibrillation waves. In the absence of conduction abnormalities, the QRS complexes should appear narrow ( $<120$  milliseconds).

The patient's pulse will feel irregular, and auscultation of the heart will reveal an irregularly irregular rhythm.

Atrial fibrillation is often paroxysmal in nature, and patients who experience palpitations should be monitored in the emergency department on telemetry to evaluate for this arrhythmia.

It is important to distinguish atrial fibrillation from other tachycardias such as supraventricular tachycardia, atrial flutter, or multifocal atrial tachycardia.

## Management

Patients who are hypotensive, altered, or otherwise unstable should be treated with synchronized cardioversion.

Treatment in the emergency department is generally focused on rate control for atrial fibrillation with RVR. Options for rate control include diltiazem, a calcium channel blocker, or metoprolol, a  $\beta$ -blocker. Both of these agents should be used with caution in the hypotensive patient.

Long-term therapy will dictate whether rate or rhythm control is best for the patient. Both seem to have similar rates of mortality and serious morbidity. Rhythm control is often preferred for symptom control, younger patient age, and irreversible structural and electrical remodeling that occurs with long-standing persistent atrial fibrillation.

Some agents such as amiodarone and digoxin function as both rate and rhythm-controlling agents.

If the onset of atrial fibrillation is >24 hours or undetermined in a patient who is not anticoagulated, chemically converting them to normal sinus rhythm places patient to an increased risk for embolic complications such as cerebrovascular accident or mesenteric ischemia.

## 6. ATRIOVENTRICULAR BLOCK

Julie Rice, Rodica Retezar



FIGURE 1. First-degree AV block. (From Lilly LS. *Pathophysiology of Heart Disease*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)



FIGURE 2. Mobitz type I, second-degree AV block. (From Lilly LS. *Pathophysiology of Heart Disease*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)



FIGURE 3. Mobitz type II, second-degree AV block. (From Lilly LS. *Pathophysiology of Heart Disease*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)



FIGURE 4. Third-degree AV block. (From Lilly LS. *Pathophysiology of Heart Disease*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Symptoms of atrioventricular (AV) block are due to hypoperfusion and/or congestive heart failure caused by bradydysrhythmia. They include dizziness, altered mental status, chest pain, shortness of breath, and syncope.



AV blocks can be precipitated by most cardiac medications including  $\beta$ -blockers, calcium channel blockers, and digitalis; by acute myocardial infarction; and by electrolyte abnormalities such as hyperkalemia and hypermagnesemia. Cardiomyopathy related to connective tissue disorders, infective endocarditis, and myocarditis can also lead to AV blocks. AV blocks are classified as first-, second-, and third-degree.

## Diagnosis

Electrocardiogram and rhythm strip provide the diagnosis and type of AV block.

First-degree AV block demonstrates a prolonged PR interval ( $>0.02$  seconds) with all P waves conducted to form QRS complexes ([Fig. 1](#)).

Second-degree AV block is divided into two types. Mobitz type I block has a progressively increased PR interval over several beats followed by an occasional nonconducted P wave ([Fig. 2](#)). This block is sometimes associated with an acute inferior myocardial infarction. Mobitz type II block has a constant PR interval with an occasional dropped beat ([Fig. 3](#)). High-grade AV block occurs when two or more consecutive P waves are not conducted. Mobitz II block can be seen during an acute anterior myocardial infarction and carries a worse prognosis than the Mobitz I variant.

Third-degree AV block is characterized by no P waves conducted through the AV node. This rhythm demonstrates constant P–P intervals from the SA node and constant R–R intervals from an escape pacemaker ([Fig. 4](#)).

## Management

For any AV nodal block, treat all electrolyte abnormalities and withhold all medications that slow conduction through the AV node. Evaluate for acute myocardial infarction and possible infectious etiologies.

For first-degree and Mobitz type I AV blocks, treatment is only necessary if the patient is symptomatic. Patients without symptoms can be discharged to follow up with a cardiologist. Patients who are symptomatic should be observed or admitted to assess the need for cardiac pacing, especially if the block is associated with a congestive heart failure. Atropine can be used to

transiently increase AV nodal conduction.

Second-degree Mobitz type II and third-degree AV blocks require temporary cardiac pacing if the patient is symptomatic or unstable. Pharmacologic treatments that can be used as a bridge to pacing are atropine, epinephrine, or dopamine infusion. Transcutaneous pacing may be used as a temporizing measure until transvenous pacing can be performed.

## 7. CONGESTIVE HEART FAILURE

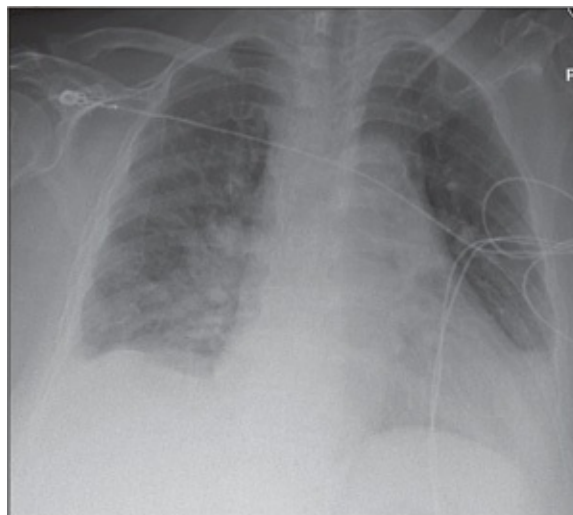
Sharon Bord, Hardin Pantle



**FIGURE 1.** Jugular venous distension.



**FIGURE 2.** Pitting lower extremity edema.



**FIGURE 3.** CXR with cardiomegaly, cephalization, and pleural effusion consistent with congestive heart failure.

**Clinical Presentation**

Patients typically present with dyspnea, orthopnea, and paroxysmal nocturnal dyspnea.

Pitting edema of lower extremities and elevated jugular venous distention may also be present.

Heart failure exacerbations are often caused by excess consumption of salt or fluid, medication noncompliance, or worsening heart function due to myocardial infarction or progressive valvular disease.

Other potential causes include alcohol or cocaine use and peri- or postpartum cardiomyopathy.

## Diagnosis

Pulmonary exam will reveal crackles or rales.

Classic findings on chest radiograph include pulmonary vascular congestion, cephalization, and pleural effusions.

Congestive heart failure (CHF) is more likely if patient has a past medical history of heart failure, atrial fibrillation, complains of paroxysmal nocturnal dyspnea, or orthopnea and has an S<sub>3</sub> present on auscultation of heart.

In equivocal cases, the diagnosis of CHF may be supported by an elevated pro-brain natriuretic peptide or point of care ultrasound demonstrating B lines in the lungs as well as plethora of the inferior vena cava.

An echocardiogram can also be performed to evaluate the patient's systolic function and ejection fraction.

## Management

Supplemental oxygen and ventilator support should be provided as needed.

Noninvasive positive pressure ventilation can be very beneficial for a patient who is experiencing pulmonary vascular congestion.

Blood pressure reduction is of utmost importance to diminish the stress that is placed on the heart. Nitroglycerin should be administered for systolic blood pressure >160 mm Hg and diastolic blood pressure >80 mm Hg to reduce mean arterial pressure. Consider starting angiotensin-converting enzyme inhibitor.

For patients with volume overload, diuretics should be employed. If patient's



home medications include furosemide, calculate patient's total daily dose of furosemide and give as a single dose intravenously (maximum 180 mg); if patient is not on furosemide as an outpatient, then furosemide 40 mg intravenous is a reasonable starting dose. Urine output should be followed to assess the patient's response.

## 8. DEEP VENOUS THROMBOSIS

Casey Wilson, Tiffany Fong



FIGURE 1. Left lower extremity DVT.

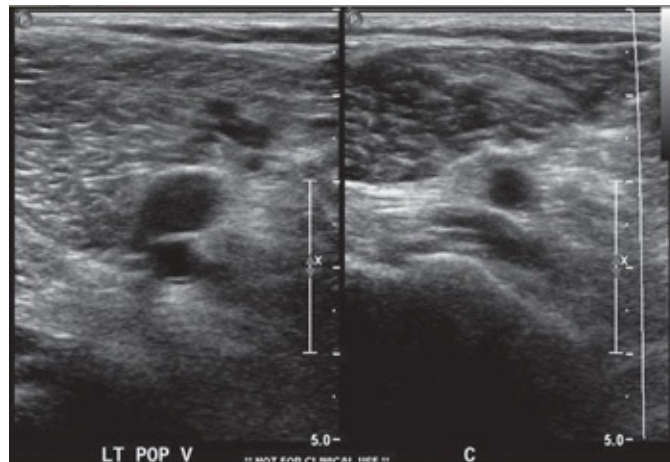


FIGURE 2. Abnormal DVT ultrasound; noncompressible popliteal vein.

### Clinical Presentation

Venous thromboembolism is a major national health problem with an overall incidence of 1 per 1,000 annually.

The most serious complication of deep vein thrombosis (DVT) is pulmonary embolism (PE), which has a mortality rate as high as 17%.

Individuals who are particularly susceptible to DVT include elderly hospitalized patients and patients who have had recent surgery, cancer, or

previous DVT.

Signs and symptoms of DVT include lower extremity swelling, pain, and erythema, but they are nonspecific and found in a wide variety of nonthrombotic disorders.

## Diagnosis

Clinical diagnosis of DVT is notoriously unreliable, and objective tests are indicated to confirm or refute the presence of this condition.

D-dimer has a 100% negative predictive value in patients who have a low pretest probability for DVT based on the Wells score. A positive D-dimer should prompt further diagnostic testing.

For patients who are at moderate or high risk for lower extremity DVT, the test of choice is compression ultrasonography of the legs. A simplified three-point compression technique can be used at the bedside, which assesses vessels with the highest turbulence and greatest risk of developing thrombus: the common femoral vein at the saphenous junction, the proximal femoral vein, and the popliteal vein. A thrombus will be identified by a noncompressible vessel; at times, echogenicity within the vessel lumen can be identified, representing thrombus. The sensitivity of ultrasound for the diagnosis of proximal DVT is 98% with specificity of 95%.

## Management

Outpatient treatment with low-molecular-weight heparin and transition to warfarin is appropriate for ambulatory patients with normal vital signs, low risk of bleeding, no renal insufficiency, and reliable follow-up.

Newer oral anticoagulants, including direct thrombin inhibitors, are an option for once daily dosing without coagulation monitoring.

Inpatient administration of unfractionated heparin is recommended if outpatient therapy is not appropriate.

Inferior vena cava filter is recommended in patients with proximal DVT or PE when there is a contraindication to or prior failure of anticoagulant therapy.

## 9. DRY GANGRENE

Hilary Schmitt, Leah Bright



**FIGURE 1.** Dry gangrene of the right leg.



FIGURE 2. Gangrene of the toes.

## Clinical Presentation

Caused by a coagulative necrosis due to gradual arterial insufficiency or occlusion

Most commonly a complication of peripheral arterial disease in patients with a history of diabetes mellitus and smoking but can also be seen in frostbite and Buerger disease

Begins at the distal periphery of extremities, spreads slowly, and is often painless

## Diagnosis

Dark, dry, necrotic tissue with clear delineation between healthy skin and eschar

Nonpalpable pulses are common. Arterial Doppler pulses and ankle–brachial indices should be obtained to assess the extent of vascular disease.

It is imperative that infections such as osteomyelitis be considered in the differential diagnosis. Plain films may be sufficient to rule out osteomyelitis, although magnetic resonance imaging is considered the gold standard.

Evidence of systemic toxicity in vital signs or laboratory studies is rare with dry gangrene and indicates a more serious diagnosis.

## Management

Consultation of vascular surgery or interventional radiology for optimization of blood flow via vascular bypass, angioplasty, or stenting is important for preventing further spread. Amputation of necrotic tissue is also required. Treatment also consists of targeting the underlying condition, for example, controlling diabetes and smoking cessation. In the absence of local and systemic signs of infection, antibiotic therapy is not indicated.

## 10. GAS GANGRENE

Hilary Fay Schmitt, Leah Bright



**FIGURE 1.** Gas gangrene of the foot.



**FIGURE 2.** Radiograph demonstrating gas in the tissues of the foot.



## Clinical Presentation

Liquefactive necrotic process, often polymicrobial, resulting in irreversible tissue destruction and associated with high mortality rate

Rapid onset (<48 hours) with pain out of proportion to what can be a relatively benign physical appearance

May result from traumatic injury, superinfection of a prior wound, or local bacterial invasion from perforated bowel

Can have similar presenting symptoms to necrotizing fasciitis, which is also a rapidly progressing surgical emergency

## Diagnosis

Physical exam may reveal edema, discoloration, subcutaneous crepitus, watery exudate, sloughing, bullae, and extreme tenderness to palpation.

Vital signs and laboratory tests show systemic signs of inflammation, such as fever and tachycardia, leukocytosis, acidosis, and end-organ dysfunction.

Plain radiographs are routine. Computed tomography scan of the affected area may be necessary to define the extent of tissue involvement.

## Management

Aggressive, early resuscitation with fluids and broad-spectrum antibiotics are important temporizing measures.

Emergent surgical consultation is required for immediate debridement and/or amputation.

Provide tetanus prophylaxis as needed.

## 11. INTRAOSSEOUS ACCESS

David Scordino, Susan Peterson



**FIGURE 1.** Interosseous access in the humeral head. (Image courtesy of Vidacare LLC. © 2014 Vidacare LLC. All rights reserved.)



**FIGURE 2.** Interosseous access in the proximal tibia. (Image courtesy of Vidacare LLC. © 2014 Vidacare LLC. All rights reserved.)

### Clinical Presentation

Intraosseous (IO) access should be considered in any patient with difficult vascular access. This can include emergent and even nonemergent cases

where peripheral access has been attempted and has been unsuccessful.

## Diagnosis

Patients may be in extremis with severe vasoconstriction and emergent need for vascular access.

Patients requiring IO access are typically in shock or respiratory or circulatory arrest.

Contraindications to IO access include fracture to the bone, previous orthopedic device at desired site, infection at insertion site, inability to identify bony structures, or recent IO insertion (last 24 hours) at desired site. Site selection is based on patient's anatomy and size, ability to locate landmarks, presenting condition, and clinical judgment. The highest success rate is in the proximal tibia due to the superficial landmarks that are easy to palpate. The humerus does have advantages over the tibial sites with higher flow rates of over 5 L/h versus 1 L/h.

## Management

### Insertion sites

Proximal tibia: 2 cm medial to the tibial tuberosity on the flat portion of the tibia. If the tibial tuberosity is not palpable, then go 2 cm distal to the inferior pole of the patella on the same flat portion of the tibia. Insert the needle at 90° angle to the bone.

Distal tibia: Site is 3 cm proximal from the medial malleolus in the flat center of the tibia.

Humeral head: Internally rotate the humerus by placing the arm across the torso. Palpate the prominent greater tuberosity, which is approximately 1 cm above the surgical neck. The IO needle should be directed 45° downward and 45° toward the patient.

Postinsertion: Remove stylet and attempt aspiration of bone marrow that will verify correct location, although there might not be marrow aspirate in all placements that are in the correct location.

Patients who are awake and are responsive to pain may require administration of preservative free 2% lidocaine (cardiac lidocaine). In adults, 40 mg of

lidocaine (2 mL) should be slowly infused over 2 minutes (in children, infuse 0.5 mg/kg to a maximum of 40 mg) followed by a flush with 10 mL of normal saline. A second dose of half the original dose can be infused over another 2 minutes if there is any pain after the flush.

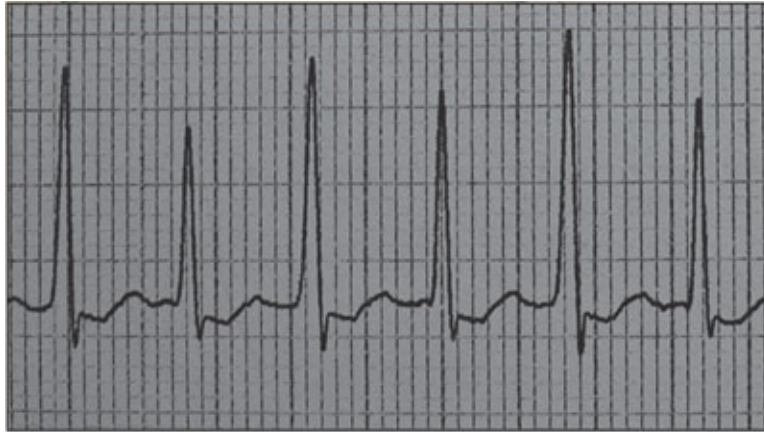
Secure the IO and attach a primed intravenous line.

Fluids and medications through the IO act centrally but must be infused under pressure with a pressure bag.

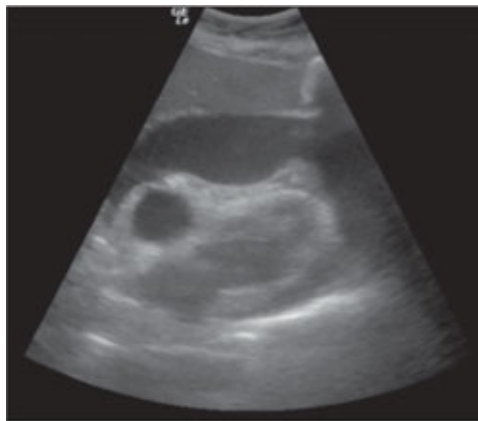
Possible complications include compartment syndrome, extravasation of fluid, fracture, bleeding, and infection.

## 12. PERICARDIAL EFFUSION AND TAMPONADE

Shaughn Keating, Tiffany Fong



**FIGURE 1.** Electrical alternans ECG. (Courtesy of Dr. Christopher Ross.)



**FIGURE 2.** Pericardial tamponade showing RV collapses. (Courtesy of Dr. John Bailitz.)

### Clinical Presentation

In pericardial effusion, there is no single sensitive/specific symptom: Patients can be asymptomatic and eventually can become dyspneic, fatigue, or develop edema.

Symptoms progress rapidly when pericardial effusion occurs acutely.

The most sensitive sign of cardiac tamponade is dyspnea.

Differential diagnosis includes acute pericarditis (viral, bacterial, tuberculous, idiopathic), autoimmune, postmyocardial infarction, postcardiac surgery, trauma, malignant, renal failure/uremia, myxedema, aortic dissection extending into pericardium, and drug-related.

## Diagnosis

Identify patients with pericardial tamponade/hemodynamic instability, who will require urgent intervention.

Classic Beck's triad of hypotension, muffled heart sounds, and jugular vein distention is uncommon.

Most sensitive sign: pulsus paradoxus  $>10$  mm Hg (also seen in pulmonary edema, hypovolemic shock, and chronic obstructive pulmonary disease)

Electrocardiogram (ECG) may show sinus tachycardia, low voltage, or electrical alternans (highly specific).

Echocardiography is test of choice. Right ventricular (RV) and right arterial diastolic collapse are specific for tamponade, but most sensitive is presence of effusion in a hypotensive patient.

## Management

Cardiac tamponade requires emergent drainage.

In patients who are too unstable to be transported to the operating room, ultrasound guided pericardiocentesis should be performed in the ED.

Most large effusions without tamponade can be managed noninvasively by treating underlying disease, but bacterial infections need to be drained surgically.

Pressors are often unhelpful because endogenous catecholamines are at a maximum.



## 13. PERICARDIOCENTESISM

Karolina Paziana, Matthew DeAugustinis



FIGURE 1. Subxiphoid approach to pericardiocentesis.

### Clinical Presentation

Emergent pericardiocentesis is performed for cardiac tamponade.

Increasing intrapericardial pressure impairs right heart filling, resulting in decreased cardiac output.

As little as 50 mL of rapidly accumulating fluid may lead to cardiovascular collapse.

### Diagnosis

Bedside ultrasonography demonstrating pericardial fluid and right ventricular (RV) or right atrial collapse is diagnostic.

Less than a third of patients present with Beck's triad of jugular venous distension, distant heart sounds, and hypotension.

Common signs include dyspnea, tachycardia, arrhythmia, pulsus paradoxus, Kussmaul's sign (increased jugular venous pressure on inspiration), electrical alternans, and low-voltage QRS.



## Management

18G spinal needle and 60 mL syringe (or largest available); commercial pericardiocentesis kits are available with all components required to perform pericardiocentesis including malleable catheter that can be left in the patient to allow continuous drainage.

Ultrasound guidance is preferred as it will dictate the best anatomical approach to drainage; if unavailable, may attach needle to precordial lead via alligator forceps, advance until fluid is aspirated, and withdraw if there is ST elevation

Subxiphoid: Insert needle below xiphoid process at 45° to skin, aiming toward left shoulder, aspirating as you advance.

Parasternal: Insert needle at parasternal border in 5th intercostal space, perpendicular to chest wall.

Apical: Insert needle one intercostal space below and 1 cm lateral to point of maximal impulse, at 45° to skin aiming toward right shoulder.

## 14. PERICARDITIS

Bachar Hamade, Jamil D. Bayram



FIGURE 1. Pericarditis.

### Clinical Presentation

The pericardium consists of two layers: visceral (epicardium) and parietal pericardium.

Pericarditis is the inflammation of the pericardium and occurs in 5% of patients who present to the emergency department with noncoronary chest pain and 1% of those who present with ST-segment elevation.

Presents acutely with severe sharp and progressive pleuritic chest pain, worse in the supine position and relieved in the sitting position, with possible radiation to the neck, arms, and left shoulder; fever and malaise are not uncommon.

Causes are multiple: idiopathic and infectious (with viral, the most common—90%), immune-mediated, myocardial infarction (1 to 3 days post-event), radiation, postoperative, malignancy, metabolic, aortic dissection, and pharmacologic.

### Diagnosis

Thorough history and physical exam are crucial. Pericardial rub (present in

35% to 85% of patients) on cardiac auscultation is pathognomonic. Laboratory findings may show leukocytosis, an elevated C-reactive protein, and sedimentation rate. Cardiac enzymes are elevated with myocardial involvement, and serologic studies are rarely of clinical relevance. Four ECG stages over several weeks (60% of patients): stage 1 (80% of patients)—diffuse ST-segment elevations and PR-segment depression in leads I, II, III, and aVF; stage 2—ST segments become isoelectric and T waves flatten; stage 3—diffuse symmetric T-wave inversion; and stage 4—normalization. Chest radiograph may show cardiomegaly when more than 200 mL of pericardial effusion is present. Echocardiography is used for detection of pericardial effusion if present.

## Management

Natural history of idiopathic or viral pericarditis is commonly favorable and management is largely supportive. Treatment consists of nonsteroidal antiinflammatory drugs (NSAIDs). A full-dose NSAID should be used (aspirin, 2 to 4 g/d; ibuprofen 1,200 to 1,800 mg/d; indomethacin 75 to 150 mg/d). Treatment should last 7 to 14 days. Aspirin is preferred if cause is myocardial infarction. Colchicine may be added if there is no response to NSAIDs.

Outpatient therapy is safe if there is no evidence of myopericarditis, trauma, immunosuppression, and in the absence of large effusion or tamponade because these are considered poor prognostic factors.

In cases with specific underlying causes, treatment should be directed toward the etiology. For example, antimicrobials for tuberculous or suppurative pericarditis, or hemodialysis for uremic pericarditis.

Complications include recurrent pericarditis in 30% of patients (consider steroids), pericardial effusion and tamponade (pericardiocentesis), and constrictive pericarditis (medical therapy plus surgical pericardiectomy).

## 15. PHLEGMASIA ALBA DOLENS AND PHLEGMASIA CERULEA DOLENS

Michael R. Ehmann, Donald Alves



**FIGURE 1.** Phlegmasia cerulea dolens. (From Mumoli N, Invernizzi C, Luschi R, et al. Phlegmasia cerulea dolens. *Circulation*. 2012;125(8):1056–1057.)

### Clinical Presentation

Phlegmasia alba dolens (PAD), “painful white edema,” is a deep venous system occlusion with a patent superficial venous system. Phlegmasia cerulea dolens (PCD), “painful blue edema,” is due to total occlusion of extremity venous outflow, including superficial veins.

PAD and PCD are a continuum of deep vein thrombosis (DVT) and if they progress, may develop venous gangrene.

Acute onset of severe pain, edema, and color change

Risk factors: hypercoagulability of malignancy, pregnancy, femoral vein catheterization, surgery, heart failure, May-Thurner syndrome

## Diagnosis

Clinical diagnosis of severe leg pain with swelling, cyanosis, edema, venous gangrene, compartment syndrome, and finally arterial compromise with subsequent circulatory collapse and shock

Confirmation with Doppler ultrasonography

May be associated with shock physiology

## Management

Definitive therapy is venous thrombectomy (interventional radiology or vascular surgery).

Consider systemic thrombolysis versus catheter-directed thrombolysis.

Initiate anticoagulation with heparin (either unfractionated or low-molecular-weight) and limb elevation promptly. However, outcomes poor with these interventions alone

Monitor closely for pulmonary embolism and associated compartment syndromes of affected limb.

Prognosis of PCD poor (20% to 50% amputation; 25% to 40% mortality)

## 16. SUPERIOR VENA CAVA SYNDROME

Hilary Fay Schmitt, Linda Regan



**FIGURE 1.** Prominent chest wall veins in a patient with superior vena cava syndrome.

### Clinical Presentation

A constellation of effects from venous congestion at the level of the superior vena cava (SVC) due to a blockage of blood return from the upper body to the heart

Blood flow obstruction is most commonly caused by external compression from the mass effect of a tumor, but it can also be caused by thrombosis within the SVC.

The most common etiologies leading to compression by a mass are lung cancer and lymphoma, although up to 40% of cases are caused by thrombosis due to intravenous devices.

### Diagnosis



Elements of the patient's past medical history that would predispose them to cancer, a known history of cancer or venous thrombus, can be diagnostic clues.

Common symptoms include swelling or flushing of the face and arms that typically develops over the course of weeks as well as cough or dyspnea. Less common symptoms can include a change in voice, headache, dizziness, or other neurologic complaints.

Rare but serious complications that would require emergent intervention include respiratory compromise (airway compression) and increased intracranial pressure (headache, syncope, vision problems, altered mental status).

Physical exam findings can include the following: swelling of the face and arms, deeper hue to the skin due to venous congestion, distention of neck and check wall veins.

Although chest x-ray may reveal some masses, computed tomography scan of the chest with intravenous contrast is a more sensitive modality for detecting both intra- and extravascular causes of SVC syndrome.

## Management

Immediate temporizing measures include elevating the head of the bed and administering supplemental oxygen.

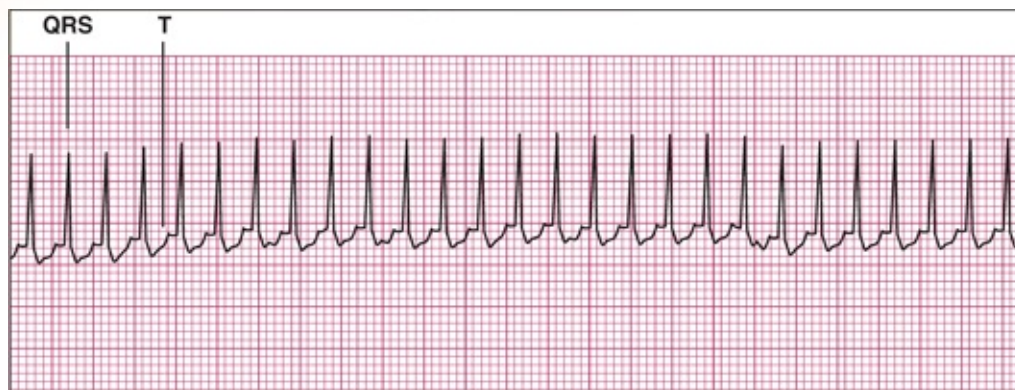
Consultation with an oncologist for urgent radiation therapy should be considered for respiratory distress or signs or neurologic compromise related to increased intracranial pressure.

SVC syndrome caused by thrombosis may be improved with removal of indwelling intravascular catheters or with administration of thrombolytics.



## 17. SUPRAVENTRICULAR TACHYCARDIA

Susan Peterson, David Scordino



**FIGURE 1.** Rhythm strip demonstrating SVT. (From Bickley LS, Szilagyi P. *Bates' Guide to Physical Examination and History Taking*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.)

### Clinical Presentation

Rapid heart rate initiating at or above the atrioventricular node. The most common etiology is paroxysmal supraventricular tachycardia (SVT).

Patients typically present with acute onset of palpitations. They may also experience dizziness, chest pain, shortness of breath, and sweating.

Prevalence is 2.25/1,000 persons, and the incidence is 35/100,000 person-years.

The mechanism is typically secondary to reentry or automaticity.

### Diagnosis

Diagnosis can be made with a 12-lead electrocardiogram (ECG).

ECG classically demonstrates a narrow complex regular tachycardia with ventricular rate of 150 to 200 bpm. P waves are often in the QRS complex or very close to it, making there appear to be abnormal or absence of P waves.

A rhythm strip may be helpful with the paper speed increased to determine if there is a presence of P waves.

Patients with antidromic atrioventricular reentry tachycardia may have a wide

complex QRS.

## Management

Hemodynamic stability must be immediately assessed, and if compromised, then synchronized cardioversion should be performed immediately.

Vagal maneuvers such as carotid massage (unilateral after auscultation for carotid bruits), ice to the face, breath holding, or asking the patient to bear down to Valsalva can be attempted initially.

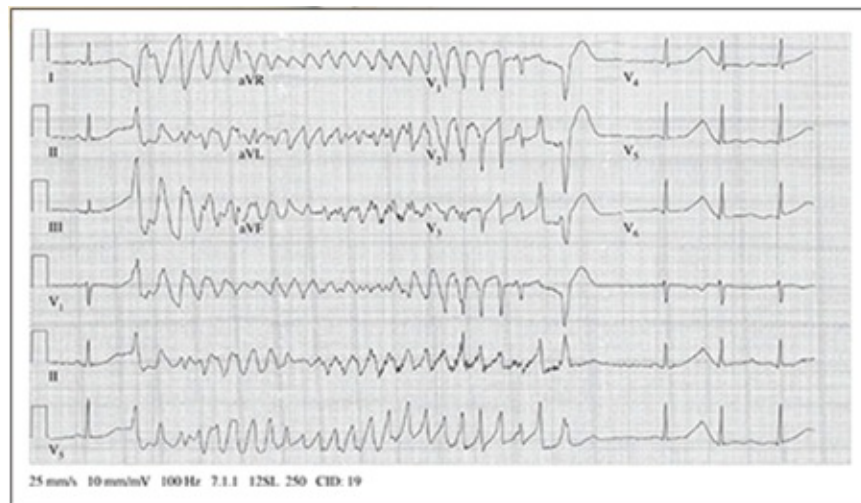
Adenosine 6 mg followed by 12 mg if not successful. A third trial of 12 mg may be considered.

Procainamide or amiodarone should be considered for those patients with SVT and suspected antidromic atrioventricular reentry tachycardia.

Synchronized cardioversion should also be considered for patients who do not respond to medications.

## 18. TORSADES DE POINTES

Caitlin M. McCord, Linda Regan



**FIGURE 1.** Torsades. (From Griffin BP. *Manual of Cardiovascular Medicine*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

A polymorphic form of ventricular tachycardia which often presents with light-headedness or syncope due to hemodynamic instability

May be self-limited or persistent and the latter can degenerate into ventricular fibrillation

Most commonly associated with prolonged QT interval but also seen in patients with structural heart disease or during acute cardiac ischemia

Etiologies for prolonged QT interval include: electrolyte abnormalities (hypokalemia, hypomagnesemia), medications that block cardiac potassium channels (some class I and III antiarrhythmics, erythromycin, phenothiazines, haloperidol, methadone, etc.), and congenital long QT syndrome.

### Diagnosis

Characteristic appearance on electrocardiography described as “torsades de pointes” (French for “twisting of the points”) in which the QRS axis swings

from positive to negative in successive beats in a single lead

Rates are often above 200 bpm.

May be preceded by R–R intervals of varying length, and then a premature ventricular contraction causing an “R on T” phenomenon

## Management

Stable patient

Give magnesium sulfate 2 g intravenous (IV) over 1 to 5 minutes followed by 1 to 2 g/h IV infusion (this may be effective even when patients have normal QT interval)

Overdrive pace at a rate of 90 to 120 bpm, which effectively shortens the QT interval

For refractory cases, give isoproterenol 2 to 10 µg/min IV infusion (carries risk of increased myocardial oxygen demand).

Correct the underlying cause typically by replacing electrolytes and stopping at-risk medications.

Unstable patient

If the patient has a pulse, perform cardioversion. If the patient is pulseless, perform defibrillation.

Prognosis is excellent if detected prior to degeneration into ventricular fibrillation.

## 19. VENOUS STASIS ULCERS

Nathan Irvin, Candice Fletcher



**FIGURE 1.** Venous stasis ulcerations are irregular and form on the medial portion of the lower leg.

### Clinical Presentation

Present in ~1% of the population, representing the most common cause of lower extremity ulceration

Results from venous incompetence secondary to immobility, valvular dysfunction, thrombosis, or phlebitis leading to an inflammatory cascade and resultant ulcer development

Risk factors include advanced age, female gender, obesity, previous leg injury, and history of deep vein thrombosis (DVT) or phlebitis.

Ulcers are often recurrent and chronic.

Complications include superinfection, osteomyelitis, and malignant conversion.

## Diagnosis

A clinical diagnosis with symptoms including dull ache and swelling relieved with elevation, and skin ulcerations that resolve and recur

Ulcers are usually shallow and irregular, located between midcalf and the ankle, and have surrounding skin discoloration and scaling.

Imaging is unnecessary, unless concerns of osteomyelitis or DVT exist, but duplex ultrasound and ankle brachial index measurements may help to rule out arterial insufficiency as cause of ulceration.

## Management

Compression therapy with compression stockings and leg elevation is mainstay of treatment.

Antibiotics are only recommended in cases of suspected superinfection, and topical creams should be avoided.

Antiplatelet agents (aspirin) as well as pentoxifylline have been shown to aid in wound healing.

Referral to wound care or vascular surgery is indicated for large or persistent nonhealing ulcers that may require specialized wound dressings, debridement, or grafting.



## 20. VENTRICULAR FIBRILLATION

Julie Rice, Julianna Jung

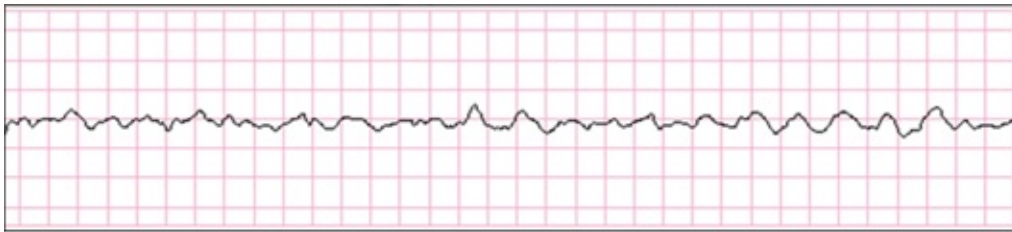


FIGURE 1. Rhythm strip of ventricular fibrillation. (Courtesy of Joseph Weber.)

### Clinical Presentation

Patient will present apneic and pulseless. Ventricular fibrillation (VF) is a rhythm often associated with sudden cardiac death.

VF is characterized by disorganized electrical activity originating from multiple foci in the ventricles. This produces uncoordinated myocardial contraction that does not generate cardiac output or a pulse.

VF is caused by many disease processes. Ischemic coronary disease is the single most common cause, and VF may be triggered by acute infarction or by arrhythmogenic foci caused by prior ischemic injury. Other etiologies include nonischemic cardiomyopathies, valvular and structural heart disease, abnormal conduction syndromes, pulmonary embolism, aortic dissection, electrical injury, drugs, electrolyte disturbances, and blunt thoracic trauma.

### Diagnosis

Check for carotid pulse in an unresponsive patient and attach cardiac monitor. Diagnosis made on monitor tracing or electrocardiogram, which will demonstrate a characteristic “zigzag” pattern without organized P or QRS complexes.

### Management



Any patient without a pulse requires immediate, continuous, and high-quality cardiopulmonary resuscitation (CPR), which has been shown to be an important determinant of survival outcomes in patients with VF.

Electrical defibrillation is the single most important treatment for VF and is essential to reorganize conduction in fibrillating myocardium. Time to defibrillation is the most important factor for patient survival, and defibrillation should always be performed within 3 minutes of cardiac arrest. Defibrillation is performed at 200 joules with a biphasic defibrillator or 360 joules with a monophasic defibrillator. Pediatric patients are defibrillated initially at 2 J/kg, with subsequent doses of 4 J/kg or more.

Defibrillation should be performed only once every 2 minutes during the course of resuscitation. “Stacked shocks” are no longer used, and there is no role for more frequent defibrillation.

Immediately after defibrillation, CPR should be continued for 2 minutes (or five 30:2 cycles) before reassessing the rhythm.

For patients with shock-refractory VF, antiarrhythmic drugs may be considered. There is no evidence that antiarrhythmics improve long-term survival outcomes for patients with VF.

If antiarrhythmics are used, amiodarone is generally the first-line agent and is given as a 300-mg intravenous bolus. If the patient remains in VF or converts to ventricular tachycardia at the next pulse check, a second 150 mg bolus may be considered. Lidocaine and procainamide may be considered as alternatives. Following return of spontaneous circulation, an antiarrhythmic infusion may be considered to reduce recurrence of ventricular dysrhythmias.

Providers must consider underlying causes of cardiac arrest and treat possible contributing factors, with particular attention to possible cardiac ischemia.

Emergent percutaneous coronary intervention is an emerging therapy for VF survivors and may improve survival outcomes for some groups.

All patients who are successfully resuscitated require intensive care unit admission for monitoring and supportive care. Therapeutic hypothermia may be considered for unconscious patients. The possible need for an automated implantable cardiac defibrillator may also be considered.

## 21. VENTRICULAR TACHYCARDIA

Christina Velasquez, Julianna Jung

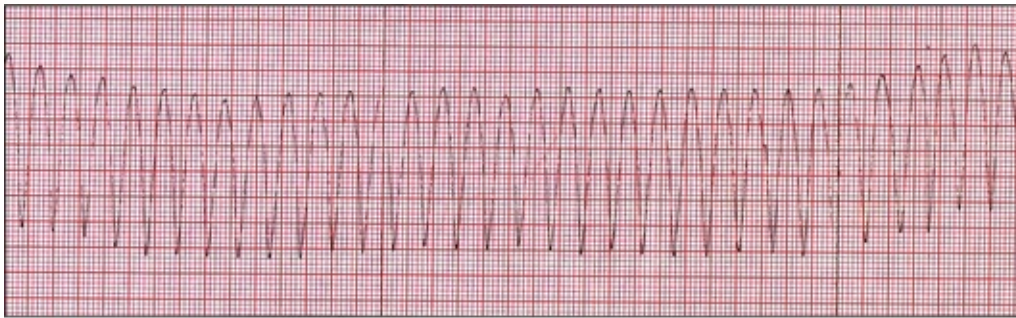


FIGURE 1. Rhythm strip of ventricular tachycardia.

### Clinical Presentation

Ventricular tachycardia (VT) occurs most commonly in the setting of ischemic heart disease; however, other etiologies include structural heart disease, abnormal conduction syndromes, or electrolyte abnormalities, and drugs. Patients in VT may be stable, unstable, or in cardiac arrest.

In patients with stable or unstable VT, signs include altered mental status, weak pulse, or rales on pulmonary exam. Symptoms include chest pain, dyspnea, palpitations, nausea, fatigue, weakness, lightheadedness, syncope, or anxiety.

### Diagnosis

Cardiac rhythm strip or 12-lead electrocardiogram (ECG) is diagnostic.

VT is any rhythm faster than 100 bpm with a wide QRS ( $>0.12$  seconds) on ECG. If QRS morphology is identical from beat to beat, VT is monomorphic. If QRS morphology varies from beat to beat, it is polymorphic.

Wide-complex tachycardia can represent monomorphic VT or supraventricular tachycardia with aberrant conduction. However, it is always safest to treat wide-complex tachycardia as VT, particularly if the patient is unstable.

Labs should focus on potassium, magnesium, calcium, and phosphate levels along with cardiac enzymes, toxicology screen, and therapeutic drug levels as clinically appropriate.

## Management

The first step in assessment is to check for a carotid pulse. Pulseless VT patients should receive immediate definitive treatment as the following. If pulse is present, assess for signs of hemodynamic instability. These include shock, altered mental status, anginal chest pain, or pulmonary edema. All pulseless patients with VT require immediate, continuous, high-quality cardiopulmonary resuscitation, which has been shown to be a key determinant of survival in cardiac arrest. Electrical defibrillation should be performed without delay in all pulseless patients. Biphasic dose is 200 J, and monophasic dose is 360 J. For shock-refractory VT, antiarrhythmic drugs may be considered, although these have not been demonstrated to improve long-term survival outcomes. Amiodarone is generally considered the first-line agent and is given as a 300-mg intravenous bolus.

In unstable VT, perform immediate synchronized cardioversion at 100 J, with prior sedation for the conscious patient when feasible. If initial cardioversion fails, additional shocks at higher doses of electricity may be given.

Defibrillation (unsynchronized cardioversion) may be required for polymorphic or irregular rhythms. For shock-refractory VT, pharmacologic intervention may be considered as the following.

For stable VT, antiarrhythmic medications should be used initially: amiodarone, procainamide, and sotalol are first-line agents. Adenosine may be considered for regular monomorphic wide-complex tachycardia in the event it represents a supraventricular rhythm with aberrant conduction. For digitalis toxicity, antidigitalis antibody is used.

## 22. WOLFF-PARKINSON-WHITE SYNDROME

Binoy Mistry, Rodney Omron

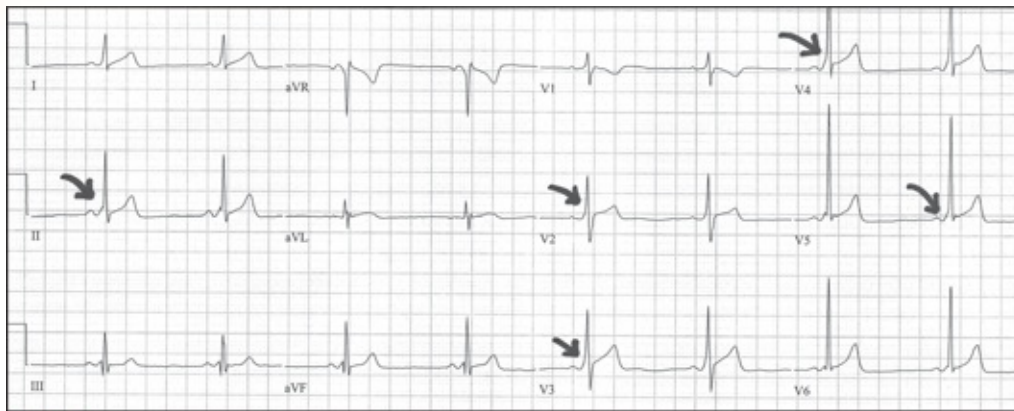


FIGURE 1. Delta waves in a patient with WPW.

### Clinical Presentation

Symptoms result from the tachyarrhythmias associated with WPW and include palpitations, light-headedness, dizziness, syncope, chest pain, and rarely, sudden cardiac death.

The etiology is an accessory pathway of myogenic tissue (e.g., Bundle of Kent) between atria and ventricles that causes electricity to bypass the atrioventricular (AV) node.

Consider WPW in patients with tachyarrhythmias with ventricular rates  $>300$  bpm and in young adults or children.

### Diagnosis

The WPW pattern is pre-excitation on electrocardiogram (ECG) without a symptomatic arrhythmia. It is associated with a short PR interval ( $<120$  milliseconds) and a delta wave.

The WPW syndrome is pre-excitation on ECG and a symptomatic arrhythmia. Orthodromic atrioventricular reentrant tachycardia (AVRT) is defined as anterograde conduction via the AV node with retrograde conduction via an



accessory pathway. This pattern will have *narrow QRS complex* (with possible negative P wave), no delta wave, and a ventricular rate of 150 to 250 bpm. Antidromic AVRT is defined as anterograde conduction via an accessory pathway and retrograde conduction via the AV node. This pattern will have a *wide QRS complex* with a delta wave and a ventricular rate of 150 to 250 bpm. Atrial fibrillation or atrial flutter associated with WPW syndrome is defined as a mix of anterograde conduction via the AV node and an accessory pathway. This pattern will have an irregularly irregular rhythm, QRS morphology and lengths that change beat to beat, and rapid AV transmission. It may have very high ventricular rates and degenerate to ventricular fibrillation.

## Management

Synchronized electric cardioversion in any hemodynamically unstable patient  
Narrow-complex supraventricular tachycardia (SVT) associated with WPW syndrome: Treat the same as SVT. Start with vagal maneuvers and adenosine.  
Wide-complex SVT associated with WPW syndrome: Treat like ventricular tachycardia. *Avoid AV nodal blocking agents* such as adenosine,  $\beta$ -blockers, or calcium channel blockers. Use procainamide (up to 17 mg/kg maximum or when the patient develops 50% widening of the QRS complex, hypotension, or the arrhythmia terminates).

Atrial fibrillation with WPW: First-line treatment is synchronized cardioversion (sedate if time allows). Second-line treatment is procainamide or ibutilide. Also *avoid AV nodal blocking agents*.

When there is anterograde conduction through an accessory pathway (wide QRS), blocking the AV node causes further conduction through the accessory pathway leading to faster ventricular rates and degeneration to ventricular fibrillation.



SECTION

G

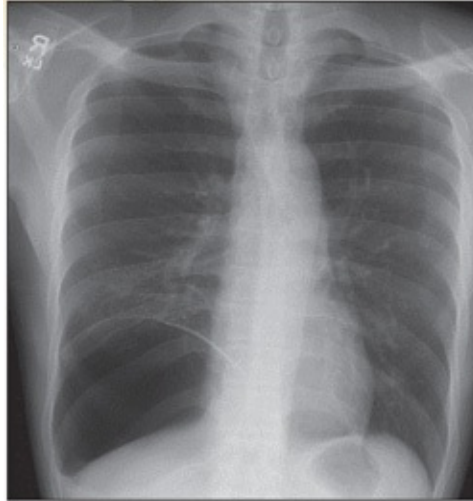
RESPIRATORY

SECTION EDITOR

Scott C. Sherman

# 1. ASTHMA

Eric J. Adkins, Andrew Krieger



**FIGURE 1.** Note the hyperinflation, flattened diaphragms, and widening of the rib spaces consistent with obstructive lung disease such as asthma. This patient also had a pulmonary bleb in the right lower lobe, which is unrelated to his obstructive lung disease.

## Clinical Presentation

Typically diagnosed during childhood but may begin later in life  
Classic symptoms include expiratory wheezing, cough, and shortness of breath.

Symptoms may be exacerbated by activity, cool air, or other inhaled substances (fumes, pollen, animal dander).

## Diagnosis

Imaging is typically normal or nondiagnostic. It may show hyperinflation with flattened diaphragms and enlarged intercostal spaces.

Physical exam demonstrates diffuse expiratory wheezing, tachycardia, or tachypnea. The expiratory phase is prolonged due to airflow obstruction. High-risk symptoms include inability to speak in complete sentences, accessory muscle use, changes in mental status, >3 emergency department visits in last year, prior hospitalization, or intensive care unit (ICU) stay for exacerbations.



Peak expiratory flow is helpful to assess the severity of symptoms. Flows <50% of predicted indicates serious airway obstruction.

## Management

Inhaled albuterol nebulizer treatments should be given immediately. In moderate to severe exacerbations, ipratropium should be used in addition to inhaled albuterol.

Prednisone 1 to 2 mg/kg/d (pediatrics) or 60 mg in adults. For impending respiratory failure, administer intravenous (IV) methylprednisolone at a dose of 60 to 80 mg every 6 to 12 hours.

Magnesium sulfate produces a bronchodilatory effect. Administer magnesium sulfate 2 g IV over 20 minutes.

Nonstandard therapies include helium–oxygen mixtures to improve airflow resistance. Additionally, ketamine or epinephrine has bronchodilator properties for severe exacerbations.

Patients who have not significantly improved after 4 to 6 hours should be considered for admission. ICU level care is indicated for patients with impending respiratory failure.

## 2. LUNG MASS

Eric J. Adkins, Lindsey Hogle



**FIGURE 1.** Chest x-ray demonstrates nodular mass in the right upper lobe.



**FIGURE 2.** CT chest slice that demonstrates the nodular mass with irregular borders.

### Clinical Presentation

A small, round object in the lung is a nodule if it is  $<3$  cm in diameter, and a lung mass if it is  $>3$  cm.

A pulmonary nodule can be either benign or malignant.

Lung nodules are usually asymptomatic. Any associated symptoms of hemoptysis, weight loss, or night sweats are concerning for a malignant process.

Many things can account for a pulmonary nodule including benign tumor,

infection, primary lung or metastatic cancer, or an inflammatory process.

## Diagnosis

A lung nodule is usually seen on a chest x-ray as an incidental finding. Computed tomography (CT) scanning should be performed as an outpatient for asymptomatic patients. CT can better characterize size, attenuation, and the type of border.

Once identified on imaging, other tests such as complete blood count, chemistry panel including calcium, and a tuberculin test can help determine if the nodule is more likely to be malignant versus benign.

Definitive diagnosis is achieved by biopsy via bronchoscopy, CT-guided needle biopsy, or by open lung biopsy.

## Management

Malignant nodules are managed by one or a combination of the following: surgery, chemotherapy, and radiation.

Benign appearing lesions do not require any treatment. A benign lesion is determined by the appearance and characteristics of the nodule, the patient's overall risk factors, and stability on imaging over a sufficient time period.

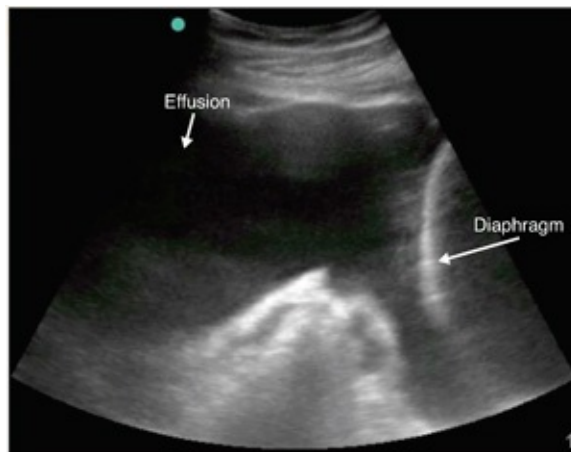
A nodule of unknown etiology requires close surveillance with serial imaging studies to assure it is not changing in size/shape.

### 3. PLEURAL EFFUSION

Sierra Beck



**FIGURE 1.** Pleural effusion on chest x-ray.



**FIGURE 2.** Ultrasound image of a pleural effusion.

#### Clinical Presentation

Small pleural effusions are often asymptomatic. As they increase in size, patients may present with cough, dyspnea, or pleuritic chest pain. Exam demonstrates absence of breath sounds over the site of the effusion and dullness to percussion. Patients may be tachypneic or hypoxic. A pleural friction rub may be present. Effusions are the result of an underlying disease process, and clinical presentation is dictated by the underlying cause. Common causes of effusion

are congestive heart failure (CHF), pneumonia, malignancy, rheumatologic disease, cirrhosis with ascites, tuberculosis, or acute atelectasis. Life-threatening causes include esophageal rupture, pulmonary embolus, aortic dissection, or hemothorax.

## Diagnosis

Effusions may be identified on upright chest x-ray when at least 200 mL of fluid are present. Lateral decubitus x-rays and ultrasound are far more sensitive and can identify as little as 5 to 10 mL of fluid. Computed tomography scan is most sensitive and may also identify underlying lung pathology contributing to the effusion, such as a mass or pulmonary embolus. If thoracentesis is performed, fluid should be sent for Gram stain and culture, cell count with differential, pH, protein, glucose, and lactate dehydrogenase (LDH).

Light's criteria may be used to determine if the effusion is a transudate or exudate. An effusion is considered exudative if one of the following conditions is met: ratio of pleural fluid protein level to serum protein level  $>0.5$ , ratio of pleural fluid LDH level to serum LDH level  $>0.6$ , or pleural fluid LDH level greater than two-thirds the upper limit of normal for serum LDH level.

## Management

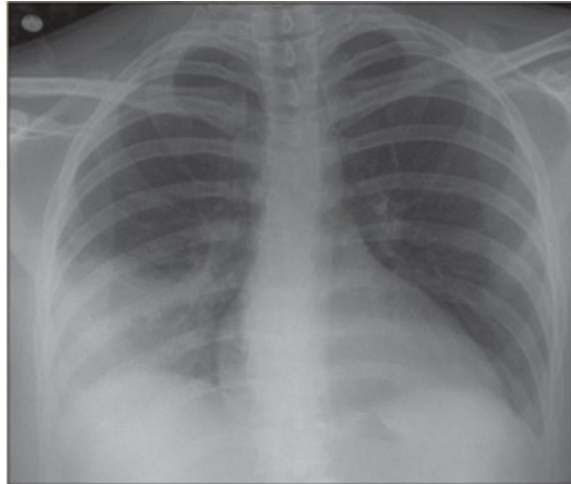
Emergent therapeutic thoracentesis should be performed in patients with large effusions and significant respiratory compromise. Diagnostic thoracentesis may be performed in the emergency setting on toxic patients or those with suspected infectious etiology where early diagnosis would assist in patient management.

Static or real-time ultrasound guidance during thoracentesis can improve success rates and decrease complications such as pneumothorax.

Management should focus on treatment of the underlying disease process (i.e., diuretics for CHF, antibiotics for pneumonia). Patients with respiratory distress or effusions of uncertain etiology will require hospital admission.

## 4. PNEUMONIA

Eric J. Adkins, Alexis Hausfeld



**FIGURE 1.** Right lower lobe pneumonia. Note the crisp right heart border. A right middle lobe pneumonia will obscure this and make it appear hazy and poorly defined.

### Clinical Presentation

The most common presentation includes cough with productive sputum, fever, shortness of breath, and pleuritic chest pain.

Typical pathogens, including *Streptococcus pneumoniae* and *Haemophilus influenzae*, account for 25% of cases.

Atypical pathogens, including *Legionella*, *Mycoplasma*, *Chlamydophila* spp. and viruses, may present with nonproductive cough, flulike symptoms, and upper respiratory infection symptoms including sore throat and nasal congestion.

Those at high risk include patients with lung diseases (i.e., chronic obstructive pulmonary disease), the elderly, recent hospitalization, and immunosuppressed (i.e., transplant recipients, cancer patients).

### Diagnosis

History along with a physical exam revealing bronchial breath sounds, inspiratory crackles, or dullness to percussion can be enough for clinical diagnosis.

Chest x-ray reveals segmental infiltration, lobar consolidation, and air bronchograms, whereas atypical pneumonia may have a more diffuse interstitial infiltrate.

Health care–associated pneumonia (HCAP) should be considered if the patient is from a nursing home, hospitalized for >2 days within last 90 days, had dialysis within last 30 days, or need for intravenous therapy within the last 30 days.

## Management

The CURB65 score helps stratify patients into risk groups based on the following signs and symptoms: *Confusion*, *Urea* >7 mmol/L (19 mg/dL), *Respiratory rate* ≥30 breaths/min, *Blood pressure* (systolic blood pressure <90 mm Hg, diastolic blood pressure ≤60 mm Hg), or *age* ≥65 years. If the patient demonstrates two or more of these symptoms, hospital treatment should be considered.

Outpatient treatment can include macrolides, tetracyclines, or fluoroquinolones.

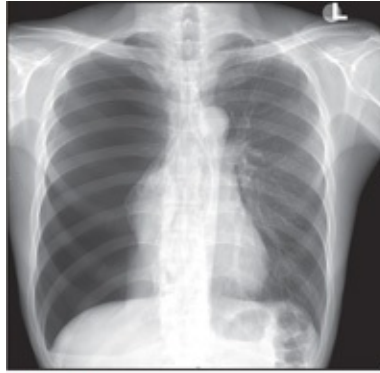
Inpatient management of community-acquired pneumonia includes ceftriaxone and azithromycin.

HCAP therapy should be guided by your local hospital antibiogram to include coverage for methicillin-resistant *Staphylococcus aureus* and *Pseudomonas* species.

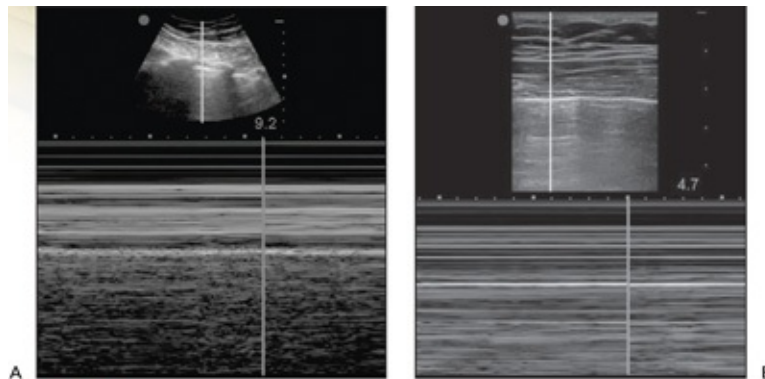


## 5. PNEUMOTHORAX

Michelle D. Lall, Natasha Demehri



**FIGURE 1.** CXR demonstrating a large right-sided pneumothorax.



**FIGURE 2.** M-mode ultrasound images. **A:** Normal "waves on a beach" indicating lung sliding. **B:** Pneumothorax indicated by absence of lung sliding. (Images courtesy of Sierra Beck, MD.)

### Clinical Presentation

**Spontaneous pneumothorax:** Patients have acute-onset shortness of breath and chest pain. They are typically thin smokers and generally healthy but may have underlying lung disease including chronic obstructive pulmonary disease, asthma, or emphysema.

**Catamenial pneumothorax:** typically 30- to 40-year-old females, commonly right-sided, occurs 2 days after onset of menstruation, and may be recurrent

**Traumatic pneumothorax:** patients with obvious chest trauma, including both penetrating and blunt trauma

**Tension pneumothorax:** dreaded complication of a closed pneumothorax; patients are typically in extremis.

## Diagnosis

On examination, patients have decreased unilateral breath sounds, hypoxia, and tachypnea and may have evidence of thoracic trauma.

Tension pneumothorax should be considered if patients are hypoxic, hypotensive, tachycardic, cyanotic, and agitated and have jugular venous distension with absent ipsilateral breath sounds.

Bedside ultrasound has the highest sensitivity and specificity and is the fastest way to diagnose a pneumothorax.

Patients may be supine or upright, and the linear probe can detect the absence of “lung sliding,” indicating a positive test. In M-mode, the “waves on a beach” sign indicates normal lung sliding and absence of this sign indicates pneumothorax.

Classically, the upright chest radiograph is used to both identify and quantify the size of the pneumothorax. It will demonstrate radiolucent absence of lung markings at the periphery, deep sulcus sign, or mediastinal shift.

If high clinical suspicion remains despite a negative ultrasound and chest x-ray (CXR), computed tomography scan can be used.

## Management

Several management strategies exist, all aimed at reduction in the size of the pneumothorax and the associated symptoms.

Needle thoracostomy should be the first treatment in any suspected tension pneumothorax.

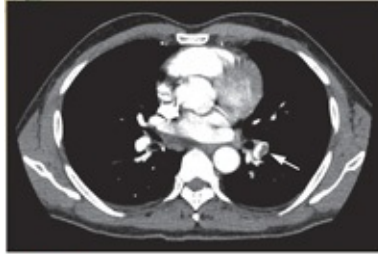
Tube thoracostomy should always follow needle thoracostomy, and it is indicated in traumatic pneumothorax and patients with respiratory distress, persistent hypoxia, and large pneumothoraces.

Small-bore, or “pig-tail” catheters, can be used in patients with uncomplicated spontaneous pneumothorax.

Supplemental oxygen, observation, and repeat chest radiographs should be obtained on stable and asymptomatic patients.

## 6. PULMONARY EMBOLISM

Jason Hine, David A. Wald



**FIGURE 1.** Chest computed tomography angiography demonstrating PE.



**FIGURE 2.** Gross specimen of clot following thrombectomy in a patient with PE. (Photo courtesy of Rob Feldman, MD.)

### Clinical Presentation

The incidence of pulmonary embolism (PE) is 1 in 500 emergency department visits.

PE continues to be a leading cause of death in the United States. Even in patients appropriately treated, the annual mortality is estimated to be between 3% and 10%.

There are a number of inherited and acquired risk factors for venous thromboembolism. Factor V Leiden thrombophilia is the most commonly identified inherited risk factor. Virchow triad (stasis, hypercoagulable state, endothelial injury) is often cited as the major acquired risk factors for PE. Shortness of breath is the most common symptom in patients with PE, occurring in up to 90% of cases. The classically described triad of pleuritic chest pain, dyspnea, and hemoptysis is much less common (<20% of cases). Unexplained tachycardia or hypoxemia should alert the clinician to the possibility of a PE.

## Diagnosis

Computed tomography (CT) pulmonary angiography is the gold standard for diagnosing PE. CT scans have the added benefit over other imaging modalities of identifying alternative causes of the patients' symptoms.

To reduce CT use and overuse, clinical decision rules such as the pulmonary embolism rule-out criteria (PERC rule), Geneva score, and Wells criteria exist to assist the physician with pretest probability.

Using physician gestalt (history, risk factor analysis, physical examination) along with the application of a clinical decision rule may help physicians risk stratify their patients and guide the clinical workup.

Ancillary testing (chest radiograph, electrocardiogram) is helpful to exclude other disease processes.

Diagnostic algorithms incorporating quantitative D-dimer testing and compression ultrasonography for deep vein thrombosis (DVT) can be used to further guide the evaluation.

Bedside echocardiography may play a role in the diagnosis of PE, particularly those who present with cardiovascular collapse. Echocardiographic findings of right ventricular dysfunction, hypokinesis, and dilation may be seen.

## Management

Anticoagulation is the cornerstone of PE management. This is usually accomplished with heparin (low-molecular-weight or unfractionated) or factor Xa inhibitors, often as a bridge to the vitamin K antagonist, warfarin.

Recently, a direct thrombin inhibitor (dabigatran) has received U.S. Food and Drug Administration approval for the long-term treatment of DVT and PE.

In the hemodynamically unstable patient, the use of thrombolytic therapy or thrombectomy is generally recommended. Controversy still exists on the use of this therapy in patients with submassive PE who are hemodynamically stable.

Inferior vena cava filter placement is another alternative for PE prevention in patients with high risk of recurrent DVT/PE for whom anticoagulation is contraindicated.

The length of therapy is dependent on the identification of a reversible

thrombotic trigger (3 to 6 months) or an idiopathic thromboembolic event (>6 months).

## 7. SARCOIDOSIS

Eric J. Adkins, Nathan Finnerty



**FIGURE 1.** Sarcoidosis. Note the bilateral lymphadenopathy on chest x-ray.

### Clinical Presentation

More common in African Americans and women; peak incidence is ages 20 to 39 years.

Pulmonary involvement is most common; symptoms may include cough, dyspnea, vague chest discomfort, or wheezing. Constitutional symptoms include fatigue, night sweats, and weight loss. Many may be asymptomatic with incidental findings on chest x-ray.

Extrapulmonary sarcoidosis may present with cutaneous (25% to 35%), ocular (25% to 80%), cardiac (25%), renal (10%), or central nervous system (10%) involvement.

### Diagnosis

Bilateral hilar lymphadenopathy on chest x-ray is the hallmark for sarcoidosis, staged from 1 to 4 based on infiltration and extent of pulmonary disease.

Erythema nodosum is a common dermatologic manifestation.  
Diagnosed by chest x-ray with compatible clinical findings and confirmed with noncaseating granulomas on biopsy  
Often a diagnosis of exclusion following workup for other possible etiologies including Gram stain and culture for acid-fast bacilli and fungi  
Serum angiotensin-converting enzyme levels are insensitive, nonspecific, and a poor therapeutic guide.

## Management

Diagnosis does not mandate treatment; however, therapy should be considered when organ function is threatened.  
Inhaled corticosteroids may improve respiratory symptoms such as cough and dyspnea.  
Oral steroids are standard initial therapy (prednisone 20 to 40 mg daily) and with demonstrated improvement in chest x-ray for patients with stage 2 and 3 disease, although there is no evidence that oral steroids alter disease progression or improve lung function.

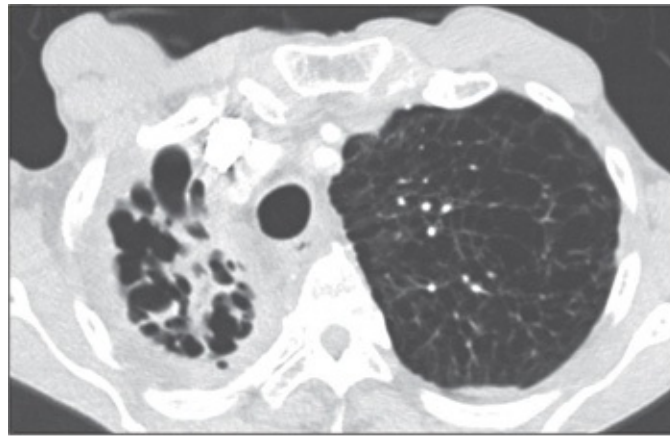


## 8. TUBERCULOSIS

Eric J. Adkins, Lindsey Hogle



**FIGURE 1.** Note the cavitation with scarring in the right upper lobe. The distal trachea and carina are also displaced to the right.



**FIGURE 2.** Chest CT slice of the same patient showing cavitation with loss of lung architecture in right upper lobe.

### Clinical Presentation

The causative organism of tuberculosis (TB) is *Mycobacterium tuberculosis*, an acid-fast bacillus that is an obligate aerobe.

TB is spread through airborne droplets from infected individuals.

Symptoms of active TB include fever, night sweats, cough, hemoptysis, and weight loss.

Primary TB is the initial infection and a normal, healthy immune system can usually keep the infection in the latent state. Secondary or reactivation TB is an active TB infection.

## Diagnosis

The purified protein derivative (PPD) skin test is used as a screening tool but will not differentiate active versus latent infection.

Sputum cultures provide a definitive diagnosis.

Chest x-ray findings of active infections include cavitary lesions that will usually be noted in the upper lobes. There may be pleural effusions, consolidations, and hilar adenopathy. Patients with latent infections will often have a normal chest x-ray.

## Management

If active infection is suspected, the patient will require respiratory isolation and hospital admission.

First-line therapy for active TB includes a multidrug regimen of isoniazid, rifampin, ethambutol, and pyrazinamide for 2 months. Several more months of treatment will usually be required based on culture results.

Other medications are available for drug-resistant cases of TB.

For individuals with a positive PPD but no clinical signs or symptoms of TB, isoniazid prophylaxis is recommended.



SECTION

H

GASTROINTESTINAL

SECTION EDITOR

Erik Nordquist

# 1. ACUTE APPENDICITIS

Basem F. Khishfe

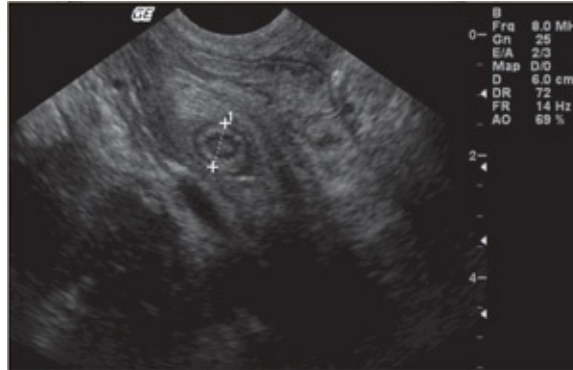


FIGURE 1. Ultrasound image displaying acute appendicitis.

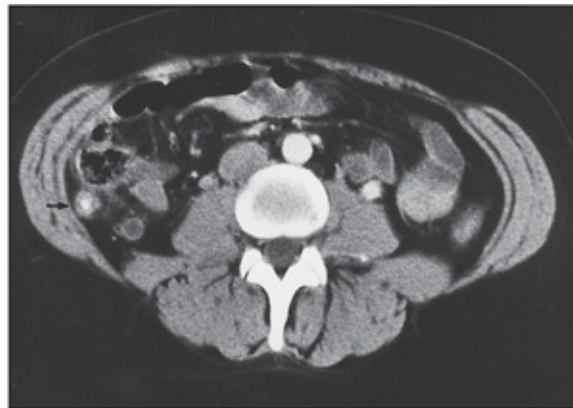


FIGURE 2. Inflamed appendix on CT (arrow).

## Clinical Presentation

All age groups are affected, but the highest incidence is in patients aged 10 to 30 years.

Common presenting symptoms include abdominal pain, anorexia, nausea and vomiting, fever, and chills.

In classic presentations, patients report vague periumbilical pain that migrates and localizes to the right lower quadrant.

Physical exam is notable for right lower quadrant abdominal tenderness; rebound and guarding may be present.

## Diagnosis

Often is clinical based on history and physical exam

Diagnostic studies may aid in patients with presentations that are not classical.

Abdominal computed tomography (CT) is the diagnostic study of choice in adult males and nonpregnant females; CT has a reported sensitivity of 97% and specificity of 98% to 100%.

Graded compression ultrasound has a reported sensitivity of 40% and specificity of 90%.

## Management

The patient should take nothing by mouth with intravenous fluids as needed.

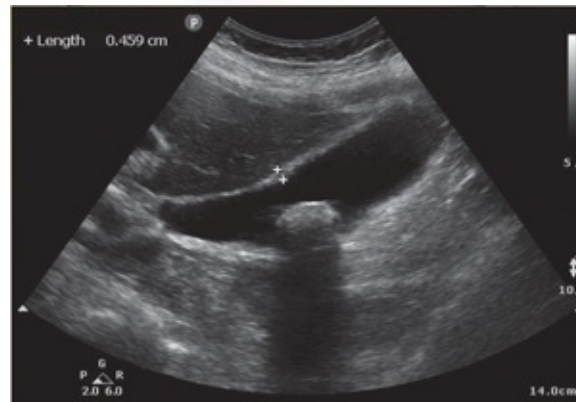
Early surgical consultation

Prophylactic antibiotics to cover gram-negative bacteria and anaerobes

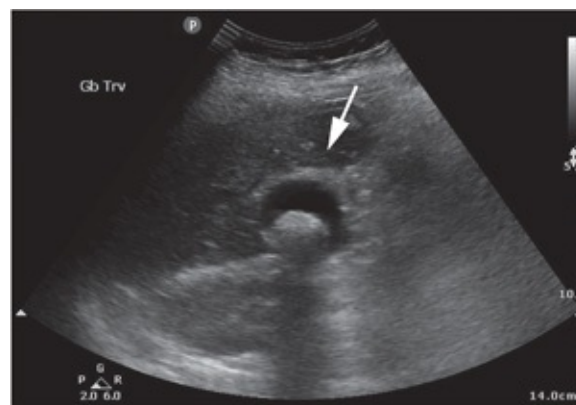
Pain control

## 2. ACUTE CHOLECYSTITIS

Damali Nakitende, Lauren M. Smith



**FIGURE 1.** Ultrasound image demonstrating a large gallstone and gallbladder wall thickening.



**FIGURE 2.** Ultrasound image demonstrating the additional finding of pericholecystic fluid (arrow).

### Clinical Presentation

Classic presentation is right upper quadrant pain, which radiates to the epigastric region and/or back.

Patients may also develop nausea, vomiting, and fever.

Physical exam is notable for right upper quadrant/epigastric tenderness.

Murphy's sign may be present.

Gallstones are the cause of acute cholecystitis in 90% of cases (calculous cholecystitis).

Acute cholecystitis can also occur in the absence of gallstones (acalculous cholecystitis).

## Diagnosis

Guided by a combination of history, physical exam, laboratory findings (i.e., elevated white blood cell, transaminases, bilirubin, etc.), and imaging

Ultrasound of the right upper quadrant is the gold standard for diagnosing acute cholecystitis.

Ultrasound findings may include a sonographic Murphy's sign, thickened gallbladder wall >3 mm, dilated common bile duct diameter >6 mm, and/or pericholecystic fluid.

## Management

The patient should take nothing by mouth with intravenous fluids as needed.

Early surgical consultation, as definitive treatment, is a cholecystectomy.

Empiric antibiotic therapy to cover gram-negative bacteria and anaerobes

Analgesics and antiemetics as needed



### 3. ACUTE PANCREATITIS

Jason Murphy

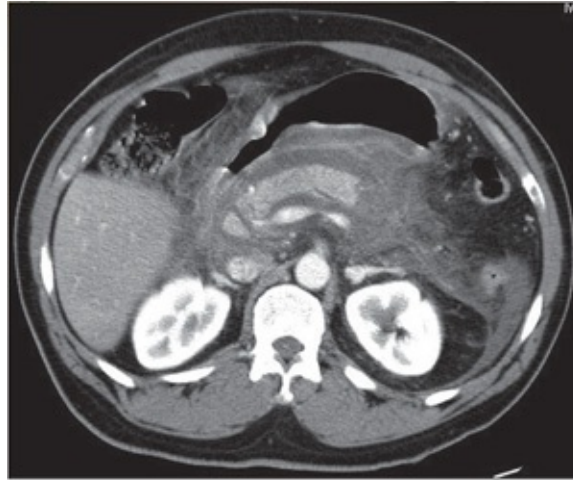


FIGURE 1. CT image of acute pancreatitis.

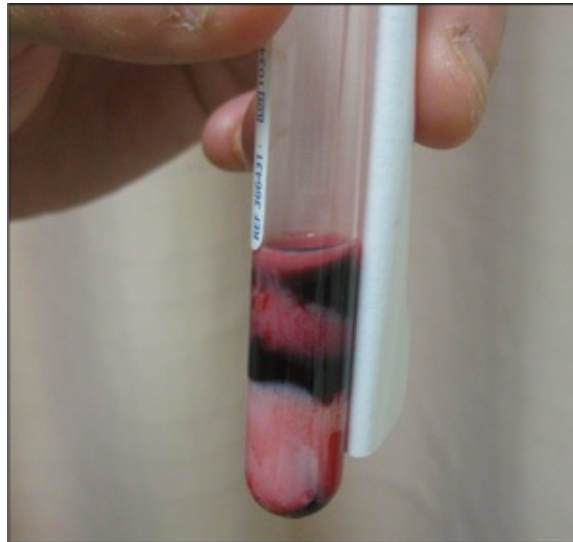


FIGURE 2. Lipemic serum sample indicating hypertriglyceridemia as the cause of the patient's acute pancreatitis.

#### Clinical Presentation

Patients typically will present with sharp or cramping epigastric pain that may radiate to the back associated with nausea and vomiting.

Occurs when some insult causes the pancreatic enzymes to become activated in the pancreas and not the gastrointestinal tract

Causes of acute pancreatitis include the following: gallstones, alcohol,

hypertriglyceridemia, post–endoscopic retrograde cholangiopancreatography (ERCP), hypercalcemia, medications, infection, toxins, and trauma. Approximately 80% of cases are caused by gallstones or excessive alcohol intake.

## Diagnosis

Must rule out other causes of upper abdominal pain, such as peptic ulcer disease and acute cholecystitis

Lipase is the most useful laboratory test, which is typically elevated 4 to 6 times the laboratory upper limit.

Abdominal ultrasound can be used to determine the presence of gallstones.

Abdominal computed tomography demonstrates inflammation of the pancreas and, if present, areas of necrosis or pseudocyst formation.

## Management

Management is largely supportive (intravenous fluids, narcotic pain medicine, and antiemetics).

Generally, patients should be kept nothing by mouth for at least 24 hours.

Treatment should also include addressing the underlying cause, for example, ERCP in pancreatitis caused by gallstones.

Third spacing can be a problem for patients with acute pancreatitis, and they may have large initial fluid requirements and may even develop hypovolemic shock.

Multiple scoring systems (Acute Physiology and Chronic Health Evaluation II, Ranson's criteria) exist to determine the severity of the presentation and to guide whether a patient should be admitted to a critical care setting.

Consider surgical or interventional radiology consultation for acute necrotizing pancreatitis, pseudocysts, or severe presentations, especially if there is evidence of abdominal compartment syndrome.

## 4. ANORECTAL ABSCESS

Bilal Khan, Rashid Kysia



FIGURE 1. Perianal abscess.



FIGURE 2. Patient undergoing incision and drainage of perianal abscess.

### Clinical Presentation

Anorectal abscesses are further divided into four subclasses based on position: perianal, ischiorectal, intersphincteric, and supralelevator. Most patients are middle-aged men.

Abscess is suggested by a history of rectal pain, tenderness, warmth, or mass with or without purulent drainage. Systemic symptoms (fever, leukocytosis) may also be present.

There may be underlying pathology, such as anal cancer or inflammatory bowel disease, particularly if the abscess is recurrent.

## Diagnosis

Perianal abscesses may be evident on external exam, appearing near the anal verge. Other anal abscesses require a digital rectal exam to feel for a fluctuant, tender mass.

Computed tomography is not routinely needed but may be required if the abscess is recurrent or the diagnosis is in question.

## Management

Perianal abscesses may be drained by emergency physicians; others require surgical consultation.

Perianal abscesses may be treated with a simple linear incision extending away from the anal verge and packed loosely.

Antibiotics are not necessary unless there are systemic symptoms. If indicated, prescribe aerobic and anaerobic coverage.

All patients should be provided surgical follow-up to ensure resolution and for treatment of possible fistulas.

## 5. ASCARIASIS

Stephanie Campbell, Rebecca Roberts



FIGURE 1. Ascariasis worm after being coughed up by patient.

### Clinical Presentation

Most common parasitic worm in the world

Patients present with initial pulmonary symptoms including cough, dyspnea, or pneumonia and can progress to include diarrhea, abdominal pain, abdominal distention, and malnutrition, as worms migrate to the gastrointestinal tract.

Loeffler syndrome is an eosinophilic pneumonia seen in infection with *Ascaris lumbricoides*.

### Diagnosis

Made with stool examination showing presence of worms or eggs

Pulmonary infiltrates on chest x-ray with eosinophilia is suggestive of infection.

### Management

Patients should be treated with an antihelminthic: mebendazole 400 mg once

or albendazole 500 mg once.

Death of large worm burden with treatment can cause bowel obstruction requiring surgical intervention.

Recurrence is common. Improved sanitation and hand washing should be discussed for prevention.

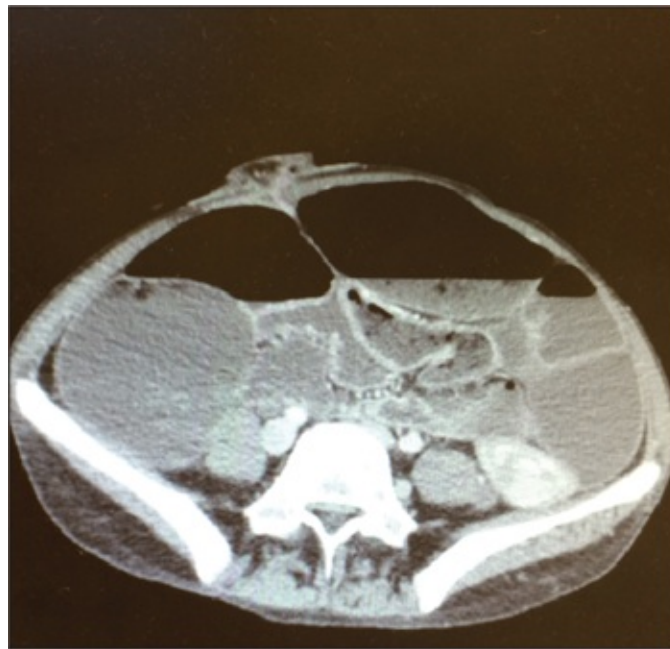


## **6. BOWEL OBSTRUCTION**

Basem F. Khishfe



**FIGURE 1.** Upright XR demonstrating air fluid levels.



**FIGURE 2.** CT image demonstrating dilated bowel and an air fluid level.

### **Clinical Presentation**

Common causes of small bowel obstruction are adhesions, hernias, and



neoplasms.

Common causes of large bowel obstruction are tumors, diverticulitis, and volvulus.

Mechanical obstruction can be partial or complete.

Crampy, intermittent, poorly localized pain associated with bilious vomiting and obstipation is typical.

## Diagnosis

Mild diffuse tenderness on exam is common.

The more proximal the obstruction, the sooner the vomiting begins.

Abdominal distention is more pronounced if the obstruction is more distal.

Plain films are often diagnostic. Computed tomography may be obtained when diagnosis remains in differential despite nondiagnostic plain films or to characterize the transition point.

Radiographic signs of small bowel obstruction are dilated loops of small bowel above the obstruction and air fluid levels.

## Management

The patient should take nothing by mouth with intravenous fluid and electrolyte replacement.

Nasogastric tube to decompress the bowel

Early surgical consultation

Pain control

## 7. CIRRHOSIS

Craig Davis



**FIGURE 1.** Abdominal distention due to ascites in a patient with cirrhosis.



**FIGURE 2.** Spider angioma. (From Smeltzer SC, Bare BG. *Textbook of Medical-Surgical Nursing*. 9th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2000.)

### Clinical Presentation

Can present as weakness, fatigue, and decreased appetite  
Patient's initial presentation may be related to a complication of underlying cirrhosis (gastrointestinal [GI] bleeding due to esophageal varices or a bleeding diathesis, abdominal pain due to spontaneous bacterial peritonitis, shortness of breath due to tense ascites, altered mental status due to hepatic

encephalopathy, or jaundice).

Ten percent to 20% of chronic alcoholics will develop cirrhosis.

Other common causes include chronic hepatitis infection and biliary cirrhosis.

## Diagnosis

Physical exam is often highly suggestive—abdominal distention, jaundice/icterus, bruising, asterixis, spider angioma, palmar erythema, muscle wasting, thinning skin, and, in males, gynecomastia and testicular atrophy.

Labs are generally nonspecific (anemia, thrombocytopenia, hyperbilirubinemia, hypoalbuminemia, coagulopathy). Aspartate aminotransaminase/alanine aminotransaminase are usually minimally elevated.

Imaging rarely necessary in emergency department (ED). Ultrasound is sensitive for ascites and can show nodularity of the liver. Computed tomography (CT) will show liver texture as well as portal hypertension and ascites.

## Management

Treatment of cirrhosis in the ED is limited to correcting fluid and electrolyte imbalances and establishing care with gastroenterologist for chronic management.

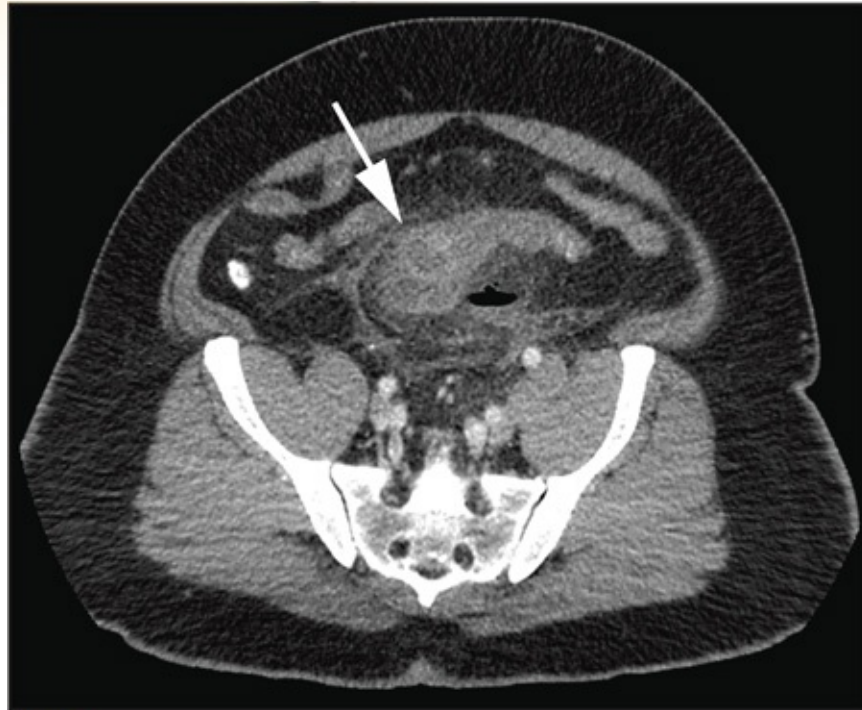
GI bleeding with coagulopathy requires fresh frozen plasma, packed red blood cells and platelets as indicated, antibiotic prophylaxis, octreotide infusion, and emergent endoscopy.

Spontaneous bacterial peritonitis often presents as altered mental status and should be diagnosed with paracentesis.

Therapeutic paracentesis should be reserved for cases in which tense ascites leads to respiratory compromise.

## 8. DIVERTICULITIS

Craig Davis



**FIGURE 1.** Complicated sigmoid diverticulitis with perforation and abscess formation.

### Clinical Presentation

Majority of cases seen in patients older than the age of 50 years.

Abdominal pain is the most common complaint.

The pain and abdominal tenderness is usually located in the left lower quadrant. However, in patients with sigmoid or right sided diverticulitis, it may be located in the suprapubic area or right lower quadrant.

Patients may also report nausea, vomiting, fever, and change in bowel habits.

### Diagnosis

Abdominal computed tomography (CT) is the preferred study for defining the extent of the disease process and possible complications (e.g., associated abscess, colonic obstruction, perforation).

## Management

Uncomplicated diverticulitis may be managed as an outpatient with oral antibiotics.

Patients with complicated diverticulitis should take nothing by mouth with intravenous fluid and electrolyte replacement as needed.

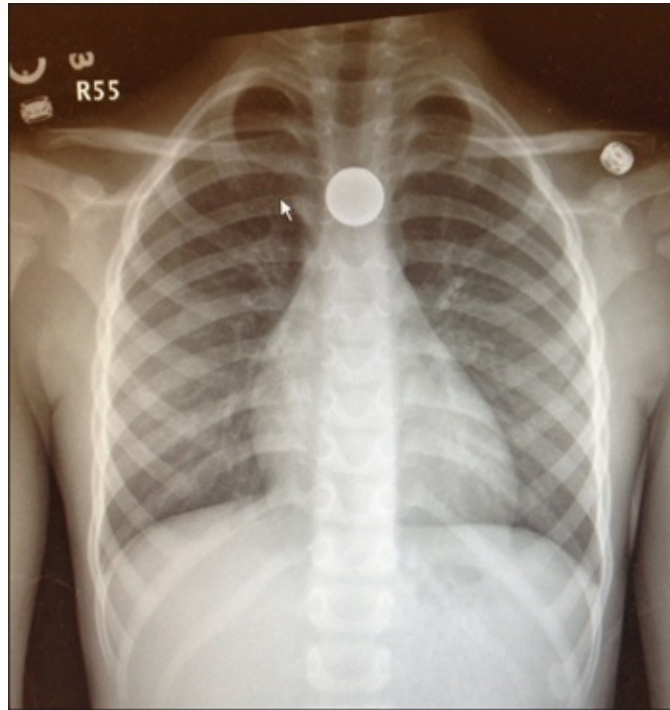
Broad-spectrum antibiotics to cover gram-negative and anaerobic bacteria

Surgical consultation for complicated diverticulitis

Percutaneous drainage may be needed for associated abscesses.

## 9. ESOPHAGEAL FOREIGN BODY

Steven Leto, Austen-Kum Chai



**FIGURE 1.** Coronal orientation of a coin in the esophagus.



**FIGURE 2.** Lateral radiograph of a coin in the esophagus.



## Clinical Presentation

Common complaints are dysphagia, drooling, vomiting, chest pain, refusal to eat, coughing, or choking with eating.

Common culprits are coins in children and food and bones in adults.

Symptoms and complications increase with prolonged duration of foreign body in esophagus.

Food impaction is associated with a high incidence of underlying esophageal pathology.

## Diagnosis

History and physical examination suggest the diagnosis.

Two-view radiographs of the neck, chest, and abdomen for radiopaque foreign bodies

Objects in the esophagus are prone to a coronal orientation.

Computed tomography without oral contrast for locating nonradiopaque objects, if clinical suspicion remains high despite unremarkable radiographs, or to assess for possible complications (e.g., esophageal perforation)

## Management

Esophageal foreign bodies require gastrointestinal consultation.

Emergent endoscopic retrieval for disk batteries, sharp-pointed objects, and complete esophageal obstruction

Coins in the esophagus may be observed 12 to 24 hours.

Consider glucagon administration to promote the passage of a food bolus.

Surgical consultation and broad spectrum antibiotics for esophageal perforation



## 10. GASTROINTESTINAL BLEEDING

Matthew Fouts, Trevor Lewis



**FIGURE 1.** Hematemesis from a child with gastritis. (From Fleisher GR, Ludwig S, Baskin MN, eds. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)



**FIGURE 2.** Bleeding duodenal ulcer. (From Britt LD, Peitzman A, Barie P, et al. *Acute Care Surgery*. Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

Clinical presentation is variable and depends on the location of bleeding, which is divided into upper and lower gastrointestinal (GI) bleeding (occurring before and after the ligament of Treitz, respectively).

Patients with upper gastrointestinal bleeding (UGIB) often present with hematemesis, melena, or epigastric abdominal pain. Brisk UGIB may present with hematochezia.

Patients with lower GI bleeding present with hematochezia in approximately two-thirds of cases and melena in one-third of cases.

Approximately 20% of patients will present with nonspecific symptoms often related to hypovolemia, such as dizziness, syncope, abdominal pain, or confusion.

## Diagnosis

Laboratory testing for suspected GI bleeding should include complete blood count, coagulation profile, liver enzymes, and type and cross. Initial hemoglobin level may not accurately reflect ongoing blood loss.

Rectal exam should be performed looking for gross blood and consideration of guaiac testing.

A nasogastric tube aspirate may provide evidence of active UGIB. However, a negative result does not rule out the possibility of hemorrhage and generally does not yield clinically useful information.

## Management

The cornerstone of management in a patient with suspected GI bleeding is adequate crystalloid resuscitation (generally 2 L intravenous fluid) and correction of coagulopathy as appropriate. Continued bleeding or instability requires prompt blood transfusion.

Proton pump inhibitor therapy has been shown to reduce rebleeding and need for surgical intervention.

Octreotide, although controversial, may be used in patients with severe uncontrolled UGIB, especially variceal bleeding.

Patients with controlled bleeding and no hemodynamic instability may be admitted for observation. Evidence of continued severe bleeding or instability should prompt admission to intensive care unit and consideration of GI, surgical, or interventional radiology consultation for definitive management.

## 11. HEMORRHOIDS

Pilar Guerrero



**FIGURE 1.** Nonthrombosed external hemorrhoid.



**FIGURE 2.** Thrombosed external hemorrhoid. (From Goodheart HP. *Goodheart's Same-Site Differential Diagnosis: A Rapid Method of Diagnosing and Treating Common Skin Disorders*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Hemorrhoids are dilated venules and arterioles of the hemorrhoidal plexuses supporting the anal canal.

Thrombosed external hemorrhoids (distal to dentate line) are commonly painful. Internal hemorrhoids (proximal to dentate line) are usually painless. Hemorrhoids are the most common cause of rectal bleeding, which is usually limited.

Risk factors include chronic constipation, frequent diarrhea, tumors, pregnancy, increased portal pressure, or increased intraabdominal pressure. Patient may present with painful or painless mass, rectal bleeding with defecation, pruritus ani, and mucous anal discharge. Hemorrhoids may be uncomplicated, thrombosed, prolapsed, strangulated, or incarcerated. More complicated presentations may include severe bleeding, abscess, thrombosis, infarction, and gangrene.

## Diagnosis

An uncomplicated hemorrhoid may appear as a skin tag, whereas a thrombosed external hemorrhoid may appear as a tender dark blue mass at the anus.

A prolapsed hemorrhoid may be visualized when the patient bears down. Internal hemorrhoids are not usually seen or palpated without the use of an anoscope.

## Management

Most uncomplicated external and internal hemorrhoids will respond to conservative therapy: warm sitz baths, analgesics, stool softeners, increased fiber, or bulk laxatives.

Topical analgesics and steroids may be helpful.

An acute (<48 to 72 hours) thrombosed hemorrhoid may be excised in the emergency department (ED) using local anesthesia. This is done by making an elliptical incision distal to anal verge on overlying skin, removing the clot, packing the wound, and applying pressure dressing for 6 to 12 hours, followed by sitz baths and a referral for hemorrhoidectomy.

Excision should not be performed in the ED for the following: children, patients who are immunocompromised, pregnant, anticoagulated, or have a coagulopathy or portal hypertension.

Surgical intervention is indicated for a strangulated or incarcerated hemorrhoid, refractory bleeding, or intractable pain.



## 12. INGUINAL HERNIA

Edward K. Lew



**FIGURE 1.** Right sided inguinal hernia.

### Clinical Presentation

Most common type of hernia

Can be indirect (hernia through inguinal ring into scrotum) or direct (through transversalis fascia in Hesselbach's triangle)

Reducible hernias are soft and easy to reduce through the hernia defect.

Incarcerated hernias are firm and usually painful and nonreducible.

Obstructive symptoms may be present. Strangulated hernias present with severe pain due to impaired blood flow. In addition, skin changes overlying the hernia may be present.

### Diagnosis

Diagnosis is based on history and physical exam.

Imaging (x-ray, computed tomography scan, ultrasound) if there is concern for obstruction or if the diagnosis is unclear

May consider lab work if concern for strangulation (i.e., white blood cell, lactate)

## Management

Reducible hernia: Patient may be discharged with outpatient surgical follow-up.

Incarcerated hernia: Attempt reduction of hernia. If the reduction is successful, the patient is appropriate for prompt outpatient surgical follow-up. However, if the reduction is unsuccessful, immediate surgical consultation is indicated.

Strangulated hernia: Do not attempt reduction. Emergent surgical consultation/intervention is indicated.

## 13. MELENA

Matthew Fouts, Trevor Lewis



**FIGURE 1.** Black tarry stool in a patient with melena.



**FIGURE 2.** Positive guaiac test indicating blood in the stool.



## Clinical Presentation

Melena is defined as the presence of dark black, tarry, and foul-smelling stool. Melena can result from as little as 150 mL to 200 mL of blood and usually appears after approximately 8 hours in the gastrointestinal tract.

Although commonly thought of as a sign of a bleed proximal to the ligament of Treitz, melena is seen in 70% of patients with upper gastrointestinal bleeding (UGIB) and 30% of patients with lower gastrointestinal bleeding. Stool that appears melenic may also result from ingestion of swallowed blood (epistaxis), bismuth subsalicylate, blueberries, black licorice, iron therapy, or lead.

## Diagnosis

Commercially available guaiac assays can help to differentiate true melena from other cause of black stool; however, false-positive tests have been reported.

Essential laboratory testing includes complete blood count, coagulation profile, liver enzymes, and a type and cross.

Nasogastric tube aspirate may help confirm UGIB; however, a negative test does not rule out blood, and the information is often not clinically useful.

## Management

The mainstay of therapy is appropriate resuscitation with crystalloid fluids, airway management as necessary, and correction of coagulopathy as indicated. Continued hemodynamic instability or bleeding after appropriate resuscitation (approximately 2 L intravenous fluids) should prompt consideration for blood transfusion.

Medical management often includes proton pump inhibitor therapy.

Patients with stable hemodynamics after appropriate resuscitation can generally be admitted to the inpatient setting for observation and further testing.

Patients with continued instability or significant coagulopathy should be monitored in an intensive care unit setting with consideration for consultation

for emergent intervention (endoscopy, angiography, surgery).

## 14. PERFORATED VISCUS

Sean Dyer, Mark Mycyk



**FIGURE 1.** Free intraperitoneal air under both diaphragms seen on chest radiograph.



**FIGURE 2.** Pneumoperitoneum (arrow) and contrast extravasation on CT.

### Clinical Presentation

Onset is usually sudden with severe localized pain that becomes generalized.

Risks include traumatic injury, caustic ingestion, peptic ulcer disease, inflammatory bowel disease, nonsteroidal antiinflammatory drugs, and alcohol.

Clinical signs include distended abdomen, rigid abdomen, and rebound tenderness.

## Diagnosis

Suspect in patients with abnormal vital signs and risk for bowel perforation  
Free air on an upright chest x-ray or lateral decubitus x-ray confirms the diagnosis.

Clinically stable patients with a nondiagnostic x-ray may require computed tomography with water-soluble contrast.

Abnormal serum tests include leukocytosis and acidosis.

## Management

Goal-directed intravenous (IV) fluid resuscitation should be initiated early.  
Broad-spectrum IV antibiotics should be administered for gram-negative and anaerobic coverage.

Surgical consultation is indicated as soon as the diagnosis is suspected.

## 15. RECTAL FOREIGN BODIES

Bilal Khan, Rashid Kysia

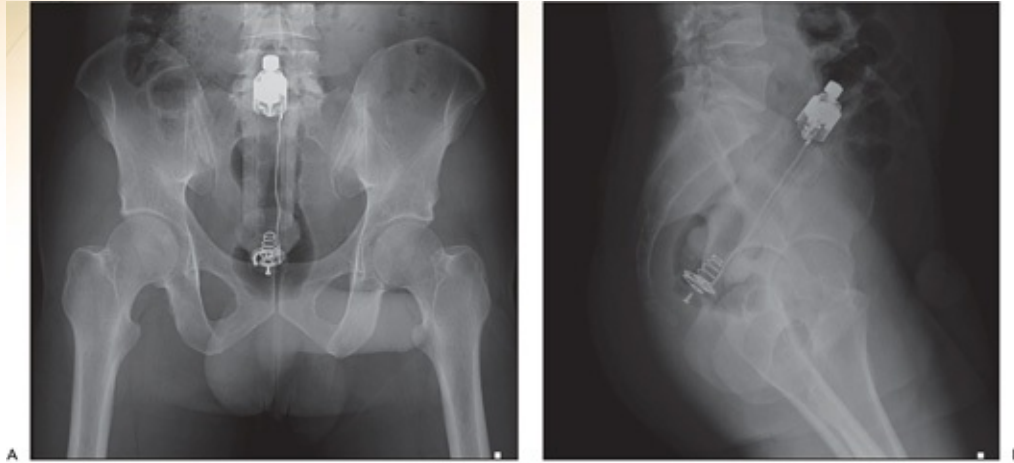


FIGURE 1. Radiographs of pelvis demonstrating a rectal foreign body. A: AP. B: Lateral.

### Clinical Presentation

Rectal foreign bodies may be inserted, intentionally or unintentionally, as a method of concealment, as means of assault, or become lodged after ingestion. A variety of objects, including those used during voluntary sexual practices, weapons, bags of illicit drugs, food items, and household items can be expected.

Although patients of both genders and any age may present, most patients are middle-aged men.

### Diagnosis

Often, but not always, diagnosed by history; embarrassment often prevents voluntarily disclosure of the foreign body.

Examine the patient for abdominal tenderness and peritoneal signs because this may indicate ischemia or perforation of the colon. Potential rectal tears or sphincter injuries must be assessed on rectal exam.

Obtain plain films to estimate size, shape, number, and location of the objects as well as to look for peritoneal and retroperitoneal free air.

### Management

Surgical services should be consulted if there is suspicion for colon perforation or ischemia, evidence of rectal or sphincter injury on exam, or for foreign bodies with sharp edges.

The patient should be placed in the lithotomy position, and the anal canal should be liberally lubricated.

Anxiolysis is essential for relaxation of the sphincter. Conscious sedation is often necessary.

The object can be grasped manually or with a variety of surgical or obstetric clamps. Gentle downward and outward traction should be applied. A vacuum effect can be broken by insertion of a Foley catheter and injection of air. One or more catheter balloons may also be insufflated and used to provide traction. If the patient is awake, bearing down may help. The use of suprapubic pressure or enemas may also be useful.

If removal in the emergency department fails or there is concern for postprocedural injury, surgery or gastroenterology should be consulted for proctoscopy and sigmoidoscopy. If per rectum removal fails, definitive management may involve laparotomy and colostomy.

## 16. UMBILICAL HERNIA

Edward K. Lew



FIGURE 1



FIGURE 2

### Clinical Presentation

Ten percent of abdominal wall hernias

Can be congenital

Can be due to increased abdominal pressure from conditions such as ascites, pregnancy, and obesity

Present with pain and/or “lump” at the umbilicus

Three classifications

Reducible: Hernia is soft, easy to reduce through hernia defect.

Incarcerated: Hernia is firm, usually painful, nonreducible.

Strangulated: severe pain, impaired blood flow, may see skin changes



overlying hernia, may have signs of obstruction

## Diagnosis

Diagnosis is based on history and physical exam.

Imaging (x-ray, computed tomography scan, ultrasound) if there is concern for obstruction or if the diagnosis is unclear

May consider lab work if concern for strangulation (i.e., white blood cell, lactate)

## Management

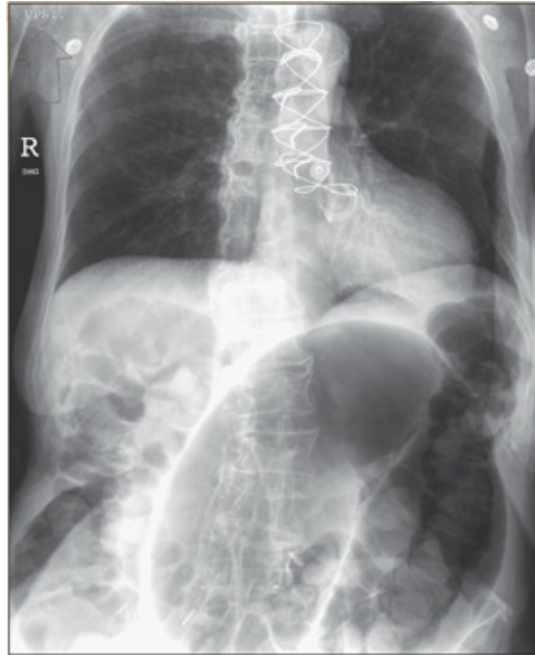
Reducible hernia: Patient may be discharged with outpatient surgical follow-up.

Incarcerated hernia: may attempt reduction of hernia. If the reduction is successful, the patient is appropriate for prompt outpatient surgical follow-up. However, if the reduction is unsuccessful, immediate surgical consultation is indicated.

Strangulated hernia: Do not attempt reduction. Emergent surgical consultation/intervention is indicated.

## 17. VOLVULUS

Helen Straus



**FIGURE 1.** Sigmoid volvulus. (From Gabrielli A, Layon AJ, Yu M. Civetta, *Taylor and Kirby's Critical Care*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.)



**FIGURE 2.** Cecal volvulus. (From Lawrence PF, Bell RM, Dayton MT, et al. *Essentials of General Surgery*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

## Clinical Presentation

Sigmoid volvulus

Most common (50%)

Risk factors include older age, pregnancy, elongated or mobile segment of bowel, psychotropic medication usage, and a high-fiber diet.

3 to 4 days of abdominal pain, abdominal distention, constipation, empty rectum on exam

Cecal volvulus

Less common (40%)

Risk factors include younger age, presence of adhesions, colonic atony, and pregnancy.

More acute onset of pain with associated abdominal distention, nausea, and vomiting

Other locations

Transverse colon, splenic flexure

## Diagnosis

“Bent inner tube” sign may be seen on abdominal x-ray in sigmoid volvulus.

“Coffee bean” sign may be seen on abdominal x-ray in cecal volvulus.

Computed tomography of the abdomen confirms diagnosis.

## Management

Sigmoid volvulus

Consult GI for flexible sigmoidoscopy to reduce the volvulus.

If endoscopy fails or the patient has peritonitis, immediate sigmoid colectomy is required.

Cecal volvulus

Consult surgery for immediate right hemicolectomy.



SECTION

I

GENITOURINARY

SECTION EDITOR

Ernest Wang

# 1. ACUTE URINARY RETENTION

Avani Desai



**FIGURE 1.** Acute urinary retention. (From Frivadossi [CC-BY-SA-3.0 (<http://creativecommons.org/licenses/by-sa/3.0>)], via Wikimedia Commons.)

## Clinical Presentation

Patients present with inability to pass urine, lower abdominal pain, and abdominal distention.

Occurs mostly in men and is the most common urologic emergency

Incidence increases with age.

The most common cause is benign prostatic hyperplasia.

Other etiologies of retention can be neurogenic, pharmacologic, traumatic, infectious, obstructive, or psychogenic.

## Diagnosis

Physical examination should include abdominal palpation, rectal exam, pelvic exam, and thorough neurologic evaluation.

Bedside ultrasound with transverse and sagittal views should be performed to estimate bladder volume after patient attempts to void.

Bladder catheterization can also be diagnostic.

Urinalysis and urine cultures should be sent.

Basic metabolic panel to assess for acute renal insufficiency if retention was prolonged

## Management

Bladder catheterization will decompress the bladder and relieve pain.

If urethral catheterization is not possible, suprapubic needle aspiration or catheterization should be performed via ultrasound guidance.

If any recent urologic procedures were performed, consult urology prior to attempting catheter insertion.

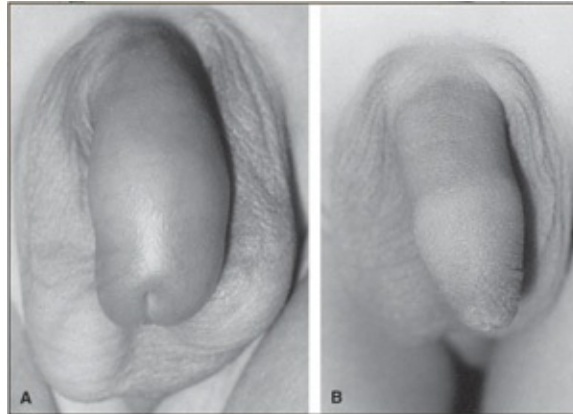
Most patients can be discharged to home with the catheter and follow up with urology for further evaluation.

Admit if the precipitating factor requires inpatient management (e.g., renal compromise, cord compression, sepsis, hematuria with clot retention).

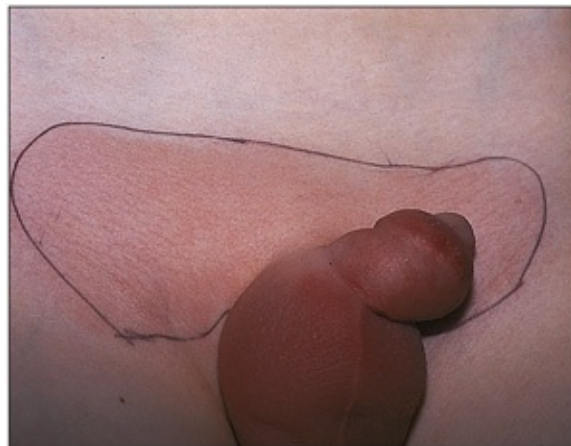


## 2. BALANOPOSTHITIS

Clare Desmond



**FIGURE 1.** **A:** Balanoposthitis—cellulitis of normal foreskin with erythema, edema, and tenderness. **B:** Normal foreskin after treatment of balanoposthitis with antibiotics and warm soaks. (From Fleisher GR, Ludwig S, eds. *Textbook of Pediatric Emergency Medicine*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)



**FIGURE 2.** Balanoposthitis with cellulitis. (Courtesy of T. Ernesto Figueroa, MD, FAAP, FACS, in Chung EK, Atkinson-McEvoy LR, Boom JA, et al, eds. *Visual Diagnosis and Treatment in Pediatrics*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Pain, redness, irritation to the foreskin and glans of the penis

Can occur in all age ranges

Fungal and bacterial causes, mostly *Candida* and *Streptococcus* species

Poor hygiene and foreskin that has difficulty retracting are often associated.



## Diagnosis

Redness, irritation, and swelling to the foreskin

Red moist macular lesion on the glans

## Management

Rule out diabetes.

Using gentle soap and water, keep the area dry and clean.

Pediatric cases are mostly bacterial; topical bacitracin may be used.

Adult cases are mostly fungal; topical clotrimazole may be used.

### 3. CONDYLOMATA ACUMINATA

Clare Desmond



FIGURE 1. Condyloma acuminata on the penile shaft.

#### Clinical Presentation

Patient will present with painless genital growths.  
Caused by the human papilloma virus (mostly subtypes 6 and 11) and commonly called *genital warts*  
High contagious sexually transmitted infection  
Incubation period of 1 to 3 months after direct contact  
Women can have increased risk of cervical carcinoma.

#### Diagnosis

Visual diagnosis, but in some cases, may require biopsy  
Soft, vegetating clusters that can appear cauliflower-like, verrucous, or lobulated  
Presents on the vulva, vagina, or cervix in females; on the penile head, shaft,

or scrotum in males; can also present perianally particularly with a history of anal intercourse

## Management

Rule out other sexually transmitted diseases and arrange follow-up.

Most people (80%) will clear the infection in 18 months.

Treatment with topical agents or ablative methods will remove the warts but not clear infection.

Common topical agents are podofilox, imiquimod, or sinecatechins.

Ablative methods include simple excision, cryosurgery, loop electrosurgical excision procedure, or laser ablation.

## 4. CONSTRICTING PENILE RING

Mark P. Kling



FIGURE 1. Bolt cutters being used to remove penile ring.

### Clinical Presentation

Teenage and adult males attempting erotic or autoerotic enhancement or for maintenance of erection

Time-dependent course: Prolonged constriction (>2 hours) leads to penile edema, vascular compromise, penile ischemia, skin necrosis, and urethral injury.

### Diagnosis

History and visualization via physical exam

Evaluate for other physical injuries to adjacent anatomy (i.e., urethral injury).

Evaluate thoroughly for extent of neurovascular compromise and anatomical involvement, so as to not extend existing injuries.

## Management

Pain control—parenteral and regional (penile block) versus local anesthesia

Clean the area around the ring; skin fragility proportional to duration of constriction

Various removal techniques have been described:

Manual removal after reduction of edema and lubricant applied (two-person Penrose method, elastic tape method)

Ring cutting (manual or electric): caution of overheating tissue or causing vascular/skin injury

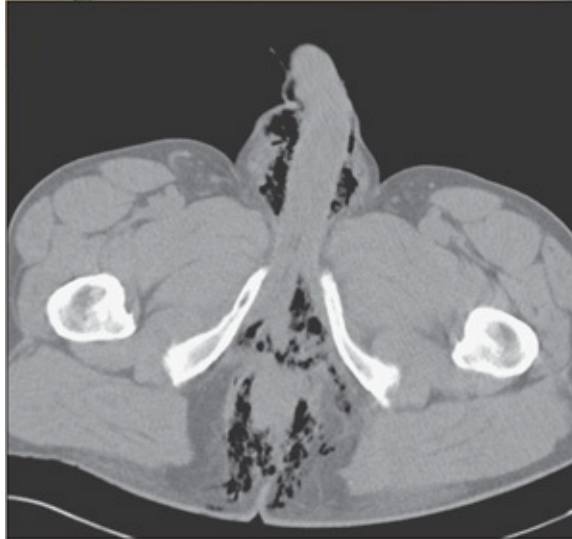
String wrap, elastic pull technique, and surgical glove techniques are also good alternatives.

Reevaluate perfusion, clean wound, antibiotic ointment.

Consult urologist for anatomical injury, inability to void, or vascular compromise.

## 5. FOURNIER GANGRENE

Rachel Spoelhof



**FIGURE 1.** CT of the pelvis showing gas in the subcutaneous tissues. (From Dunnick R, Sandler C, Newhouse J. *Textbook of Uroradiology*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)



**FIGURE 2.** Fournier gangrene in a female. (Courtesy of Paul J. Kovalcik, MD.)

### Clinical Presentation

Fournier gangrene is necrotizing fasciitis of the perineum from cutaneous, perianal, colorectal, genitourinary, or traumatic source.

Anal, rectal, or colonic sources tend to have a worse prognosis.

Most common in men. Risk factors include diabetes, alcohol abuse, and immunosuppression.

Mortality can reach up to 40% even with appropriate treatment.



## Diagnosis

Largely a clinical diagnosis; pain, redness, swelling, induration, necrosis, or perineal crepitus may be present.

Look for signs of sepsis: fever, hypotension, tachycardia, leukocytosis, and altered mental status.

Have a high index of suspicion; the physical exam may be unimpressive.

Radiographs may show soft tissue gas; ultrasound may show scrotal wall thickening, subcutaneous air, or peritesticular fluid. Testes will appear normal because their blood supply is derived from a separate source.

Consider computed tomography (CT) when diagnosis is equivocal; however, keep in mind these patients are septic and can decompensate quickly.

## Management

Immediate urologic consultation and surgical debridement is mandatory.

Infection is usually polymicrobial although anaerobes are most common.

Broad antibiotic coverage and aggressive fluid resuscitation is the mainstay of emergency department treatment.

Triple therapy antibiotic coverage is recommended. Consider antifungals for high-risk patients.



## 6. GENITAL HERPES

Pedro Cardama, Jason P. Stopyra



**FIGURE 1.** Herpes simplex on the penis. (From Craft N, Fox LP, Goldsmith LA, et al. *VisualDx: Essential Adult Dermatology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)



**FIGURE 2.** Genital herpes is a viral infection that causes blisters to form on the vulva (in women). (© Dr. P. Marrazi/Photo Researchers, Inc.)

## Clinical Presentation

Genital herpes is primarily caused by herpes simplex virus (HSV)2, but the incidence of genital herpes due to HSV-1 has increased significantly.

Transmission is due to direct contact, and the viruses are highly contagious, even when patient is asymptomatic.

Lesions present as grouped, painful vesicles with erythematous bases that later ulcerate into shallow ulcers, which may coalesce. These lesions may be located on the vulva, vagina, cervix, perineum, buttocks, penile shaft, or glans. The lesions of primary herpes can last up to 3 weeks and heal without scarring. Systemic manifestations may include fever, malaise, myalgias, headache, tender inguinal lymphadenopathy, or dysuria.

Recurrences are common but are typically less severe in number and duration than primary infections and lack systemic symptoms. They are often heralded by either tingling or numbness in the affected area.

## Diagnosis

Clinical diagnosis should be confirmed using laboratory testing such as viral culture, polymerase chain reaction, direct fluorescent antibody, Tzanck smear, and/or serology.

Consider testing for other sexually transmitted illnesses.

## Management

Genital herpes is lifelong, and patients should receive counseling and education. Using condoms does not fully protect from transmission.

Antiviral therapy of genital herpes is separated between primary and recurrent infection.

Primary: acyclovir 400 mg three times daily or 200 mg five times daily for 7 to 10 days. Alternative treatments include valacyclovir 1 g twice daily for 7 to 10 days or famciclovir 250 mg three times daily for 5 to 10 days.

Recurrent: acyclovir 400 mg orally three times a day or 800 mg orally twice a day for 5 days. Alternative treatments include famciclovir 125 mg orally twice a day for 5 days or valacyclovir 500 mg orally twice a day for 3 to 5 days.

## 7. HYDROCELE

Avani Desai



FIGURE 1. Hydrocele clinical picture.

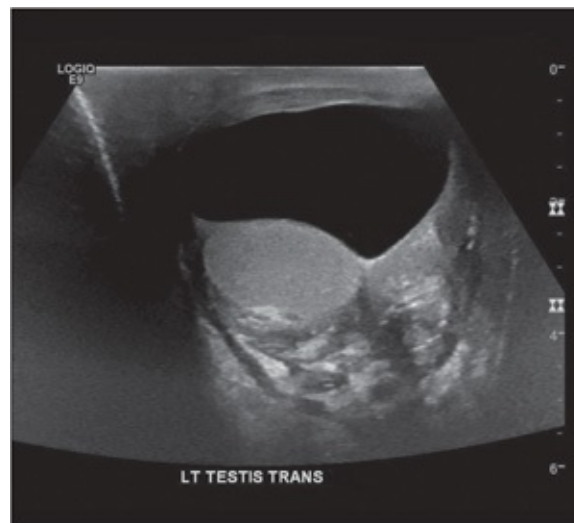


FIGURE 2. Hydrocele (bilateral) ultrasound.

### Clinical Presentation

Fluid collection between the parietal and visceral layers of the tunica vaginalis  
In infancy, hydroceles are often associated with a patent processus vaginalis and an indirect hernia.

In older children and adults, hydroceles can be idiopathic or reactive secondary to an underlying hernia, epididymitis, orchitis, trauma, neoplasm,

or torsion.

Symptoms include scrotal discomfort, swelling, and fullness.

Pain is not commonly associated with a hydrocele but can be present with an acute fluid accumulation or a significant expansion of a chronic fluid collection.

## Diagnosis

Physical examination reveals an enlarged, nontender scrotum.

Transillumination can differentiate it from a solid mass due to its cystic nature.

An ultrasound will confirm the diagnosis and is useful in detecting associated pathology.

## Management

Although usually idiopathic, the presence of a hydrocele should incite further evaluation for causative process if suspected.

If not associated with a hernia, hydroceles usually resolve spontaneously within the first year of life.

Surgical treatment should be considered if it is associated with a hernia, communicates with peritoneal space, persists from birth after age 2 years, or causes considerable discomfort.

Aspiration is ineffective due to eventual reaccumulation of fluid.

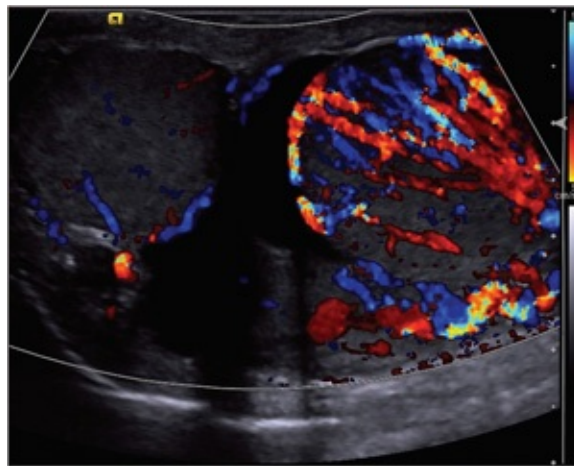
The most common treatment is surgical excision of the hydrocele sac.

## 8. ORCHITIS

Saada Zegar, Jodi Carrillo



**FIGURE 1.** Right epididymo-orchitis



**FIGURE 2.** Scrotal ultrasound showing both testicles. The left testicle and epididymis demonstrates evidence of inflammation. Notice the thickened scrotal wall, heterogeneous echogenicity of the testicle, and increased blood flow on Doppler.

### Clinical Presentation

Inflammation of the testicle usually presenting with the subacute onset of testicular pain, swelling, and tenderness

Isolated orchitis is most commonly viral in etiology, usually mumps.

Bacterial orchitis usually occurs in conjunction with epididymitis (epididymo-orchitis).



In sexually active males aged 14 to 35 years, consider sexually transmitted infection (STI), usually caused by *Neisseria gonorrhoeae* or *Chlamydia trachomatis*.

In males <14 years or >35 years, causative agent is usually *Escherichia coli*. Noninfectious causes include vasculitides and amiodarone.

## Diagnosis

Physical exam findings include testicular enlargement, induration of testicle, tenderness, and erythematous/edematous scrotal skin. Testicle should have normal lie and cremasteric reflex.

Ultrasound should be used as adjunct to rule out torsion with acute scrotum.

Ultrasound can confirm testicular inflammation and involvement of epididymis.

Urine analysis and urine culture should be sent to confirm pathogen.

For sexually active males, perform urethral culture and Gram stain or polymerase chain reaction.

## Management

Mumps orchitis resolves spontaneously in 3 to 10 days.

Supportive therapy is mainstay of treatment: bed rest, hot/cold packs, and scrotal elevation.

For sexually active males with epididymo-orchitis, empirically treat for STI (ceftriaxone/azithromycin).

For bacterial orchitis in males 35 years or older, treat with 7- to 14-day course of fluoroquinolone (ciprofloxacin).

If orchitis is caused by amiodarone, discontinuation of the medication is required.

Follow up with primary doctor or urologist.

## 9. PENILE FRACTURE

Christopher Davis, Jason P. Stopyra



FIGURE 1. Penile fracture with classic "egg-plant" deformity. (From Britt LD, Peitzman A, Barie P, et al. *Acute Care Surgery*. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

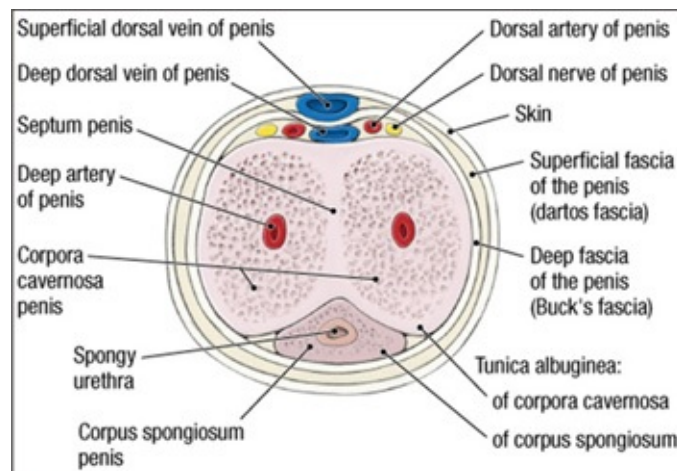


FIGURE 2. Transverse section through the body of the penis. (From Tank PW. *Grant's Dissector*. 15th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

Penile fractures result from the traumatic rupture of the tunica albuginea covering either one or both of the corpus cavernosae.

Most injuries occur as a result of vigorous sexual intercourse or self-manipulation wherein the erect penis is forcefully bent, causing a buckling injury.

Patients often report hearing "snapping" or "popping" sound followed by rapid detumescence, pain, and swelling/bruising of the penis.

Due to embarrassment, patients may present with nonsexual explanations for



their injuries.

## Diagnosis

Penile fracture is primarily a clinical diagnosis.

On exam, the penis will be flaccid, swollen, ecchymotic, and often deviated away from the side of the ruptured tunica. This is termed as *eggplant deformity*.

On examination, a hematoma can be palpated directly overlying the tear in the tunica, and it is often immobile because the skin of the penis is rolled over it.

## Management

Occult urethral injuries may be identified by checking urinalysis for presence of hematuria.

If the patient has urinary retention, a retrograde urethrogram may be necessary to look for underlying urethral injury.

Urologic consultation should be obtained urgently because surgical management is currently overwhelmingly favored over conservative, nonoperative treatment.

## 10. PHIMOSIS

Ernest Wang



**FIGURE 1.** Phimosis in a pediatric patient. (Courtesy of T. Ernesto Figueroa, MD, FAAP, FACS, in Chung EK, Boom JA, Datto GA, et al, eds. *Visual Diagnosis in Pediatrics*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Inability to retract the foreskin covering the glans penis in uncircumcised males

Physiologic disorder is present at birth and usually resolves by age 5 to 7 years. Pathologic etiologies include trauma, inflammation, or infection.

### Diagnosis

Pain or bleeding with forceful retraction

Ballooning of the foreskin during urination, dysuria, painful erections, or infection (balanoposthitis or urinary tract infection)

### Management

Outpatient urology referral

Topical steroids for 6 to 8 weeks and gentle manual retraction  
Antibiotics as indicated for secondary infection  
Circumcision rarely necessary

## 11. PRIAPISM

Larry B. Mellick



**FIGURE 1.** A: A single, large-bore, butterfly needle is placed proximally at either 2 or 3 o'clock or 9 or 10 o'clock into the corpus cavernosa, avoiding the dorsal neurovascular structures and the urethra. B: An optional second needle or catheter placed more distally on the opposite side of the penis may be used to aspirate when saline irrigation is performed. C: Successful aspiration of blood. D: Phenylephrine (1 mL of 100 to 500 mg/mL) injections are performed every 5 minutes. Use lower concentrations for children and patients with severe cardiovascular disease.

### Clinical Presentation

Involuntary, prolonged erection lasting longer than 4 hours; unrelated to sexual stimulation and not relieved by ejaculation

Ischemic (low-flow) priapism is much more common than nonischemic (high-flow). Nonischemic priapism is caused by a vascular fistula resulting in unregulated arterial inflow and is not considered an emergency.

Low-flow priapism is considered a compartment syndrome of the penis, and a timely intervention is important to preserve the sexual function of the penis.

Associated with intracavernosal medications and sickle cell disease

### Diagnosis

Nonischemic priapism is less painful and is typically associated with a history of trauma.

Testing of a new, undifferentiated priapism patient may include a complete blood count, reticulocyte count, hemoglobin electrophoresis, psychoactive medication screening, urine toxicology, penile blood gas testing, color duplex ultrasonography, and rarely penile arteriography.

## Management

Local injections with lidocaine or bupivacaine without epinephrine or a penile dorsal nerve or ring block along with systemic opiates are recommended for pain control.

Therapeutic aspiration for low-flow priapism, with or without irrigation, or intracavernous injection of a sympathomimetic agent (i.e., phenylephrine); aspiration is often recommended first because there is some evidence that the acidotic, anoxic, hypercarbic, and hypoglycemic blood decreases the effectiveness of the sympathomimetic.

## 12. RENAL STONE

Maria Coganow



FIGURE 1. Left ureterovesicular renal stone.

### Clinical Presentation

Most common in the third to fifth decade of life. Three times more common in males

Acute onset of severe unilateral flank pain. May be associated with radiation to the groin, nausea, vomiting, and diaphoresis

“Renal colic” describes the writhing, pacing, and anxiousness secondary to dilatation, stretching, and spasms of acute ureteral obstruction.

Calcium oxalate stones are the most common. Other types include struvite, uric acid, and cystine.

### Diagnosis

Urinalysis to assess for microscopic hematuria and infection

Check creatinine to assess renal insufficiency secondary to obstruction.

Noncontrast helical computed tomography (CT) is the mainstay of diagnosis (96% positive predictive value; 93% to 97% negative predictive value).

Ultrasound (US) detects hydronephrosis of larger stones but is not sensitive for midureteral stones or small stones.

Differential diagnosis includes abdominal aortic aneurysm, aortic dissection, appendicitis, pyelonephritis, cholecystitis, testicular or ovarian torsion, and

herpes zoster.

## Management

Fluid administration is indicated for dehydration or renal insufficiency.

Analgesia: Nonsteroidal anti-inflammatory drugs are the analgesic of choice because the prostaglandins promote dilation of the ureter. Intravenous opioids such as morphine and hydromorphone are also effective.

Medical expulsion therapy:  $\alpha$ -Blockers (e.g., tamsulosin) have shown to improve expulsion time by 2 to 6 days.

Patients can be discharged home if they have small ( $<6$  mm) stones with normal kidney function, uncomplicated infections, and pain controlled on oral analgesics. They should be sent with urine strainer, oral analgesics,  $\alpha$ -blockers, and urology follow-up.

Patients with large obstructing stones ( $>6$  mm), renal insufficiency, solitary or transplant kidney, signs of urosepsis, or uncontrolled pain should have urgent urology consult and require admission. Most require some type of surgical intervention, such as stent placement, percutaneous nephrostomy, or extracorporeal shockwave lithotripsy.



## 13. RHABDOMYOLYSIS

Sari Hart



**FIGURE 1.** A: Haiti earthquake building collapse and crush victim. B: "Coca-cola" colored urine and urine dip that falsely detects blood due to presence of myoglobin. (Courtesy of Heather Costello, MD.)

### Clinical Presentation

Clinical syndrome of muscle injury with release of cellular contents, particularly myoglobin, which can lead to renal injury

Causes include the following: crush or mechanical injury; excessive muscle contraction (status epilepticus or seizures, excessive exertion, delirium tremens); electrical injury (high voltage, lightning strike); heat damage (neuroleptic malignant syndrome, malignant hyperthermia, burns, heat stroke); dependency (i.e., prolonged down time in prone position); medications (especially statins); envenomation (snake, hymenoptera); metabolic myopathies (e.g., McArdles); infectious: viral myopathies (coxsackie, Epstein-Barr virus, HIV, influenza), bacterial (*Legionella*, *Salmonella*, *Shigella*)

Classic clinical triad of muscle pain, weakness, and tea-colored urine is present in <10% of cases. Up to 50% will have no myalgia nor weakness.

### Diagnosis

Myoglobinuria: dark urine, dipstick positive for blood but *no* red blood cells

on microscopic exam

Creatine kinase (CK) levels elevated >5 to 10 times upper limit of normal.

Myoglobin clears rapidly from plasma, and pigmenturia may resolve as quickly as 6 hours, but creatine kinase remains elevated for 3 to 5 days.

Risk of renal failure increases with comorbid conditions (sepsis, acidosis, dehydration) and with degree of CK elevation.

## Management

Early and aggressive volume expansion with normal saline. Monitor urine output, goal of 3 to 4 mL/kg/h.

Alkalinization of the urine: 1 to 2 amps of sodium bicarbonate in 1 L of 0.45 NaCl intravenous fluid, target urine pH >6.5. Alkalinization increases solubility of myoglobin and renal excretion. (Mannitol controversial: unclear that it offers any benefit over volume expansion)

Correct electrolyte imbalances, with particular attention to potassium and phosphorus. Monitor overall volume status to avoid volume overload.

Dialysis when indicated: refractory hyperkalemia, resistant metabolic acidosis, and acute oliguric renal failure complicated by fluid overload and congestive heart failure.

Identify and treat the underlying causes.

## 14. TESTICULAR TORSION

Martin Fedko



**FIGURE 1.** Torsion of the left testicle. Note that it is "high riding."

### Clinical Presentation

Patients usually present with abdominal, inguinal, and/or testicular pain. It presents in a bimodal age distribution, but it can occur at any age. A reasonable number occur during sleep because unilateral muscle contractions take place at this time.

Torsion usually occurs without a traumatic event. Only 4% to 8% are associated with trauma.

Torsion occurs because of an abnormal fixation of the testis within the tunica vaginalis.

### Diagnosis

Make sure the patient is standing for the exam because this will yield optimal results.

The most sensitive finding in torsion is unilateral absence of the cremasteric reflex, which occurs in up to 99% of cases.

The affected testis may be high-riding as compared to the other testis.

The affected testis is often tender and firm on exam.

## Management

Attempt detorsion using the open-book technique. The initial rotation should be 540°. The affected testis should be rotated counter clockwise if it is the right testis or clockwise if it is the left testis.

Emergent urologic consultation

Do not delay consultation for imaging.

Doppler ultrasound is the preferred imaging modality.



SECTION

J

OBSTETRICS/  
GYNECOLOGIC

SECTION EDITOR

Ernest Wang



# 1. BARTHOLIN GLAND ABSCESS

Vivian Isrow



FIGURE 1. Left Bartholin's gland abscess.

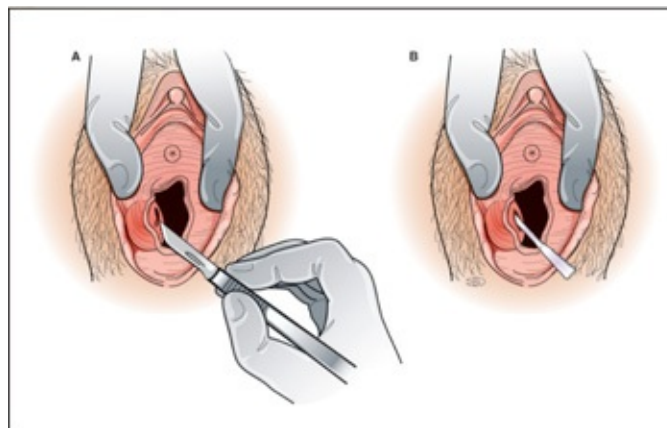


FIGURE 2. Treatment options of a Bartholin abscess. **A:** I&D of a Bartholin abscess: Make a vertical incision over the mucocutaneous junction on the vaginal mucosal side of the labia minora. **B:** Word catheter insertion: Make a 0.5-cm long stab incision into the duct or abscess, drain the cavity, insert the Word catheter as shown, and inflate the balloon with saline. (From Simon R, Ross C, Bowman SH, et al. *Cook County Manual of Emergency Procedures*. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.)

## Clinical Presentation

Bartholin glands produce the mucus secretions to provide lubrication of the vulva.

Both infection and trauma can lead to the prevention of drainage that causes abscess formation.

Infected Bartholin glands can be secondary to sexually transmitted diseases (i.e., gonorrhea and chlamydia) or can be polymicrobial.

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## Diagnosis

Bartholin glands are located at the 4 and 8 o'clock positions of the vaginal introitus.

Bartholin abscesses are typically identified as 1 to 4 cm in diameter of fluctuant, tender, erythematous swelling in the inferior labia majora. Symptoms include dyspareunia as well as pain with walking/sitting.

## Management

Warm compresses can be used.

Incision and drainage (I&D) of an abscess should be performed.

After I&D, a Word catheter (a small catheter which has a balloon tip) should be inserted and left in place for 4 weeks to allow for a new duct to form.

Antibiotics can be started based on culture results or if there is surrounding cellulitis.



## 2. BREAST INFECTION

Clare Desmond



FIGURE 1. A: Lactational breast infection. B: Large abscess was present on ultrasound which was treated by aspiration with rapid resolution. (From Harris JR, Lippman ME, Morrow M, et al, eds. Diseases of the Breast. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.)

### Clinical Presentation

Patient will present with pain, fever, and erythema to the breast area often during lactation.

History will often reveal engorgement, trouble breastfeeding, and chafed nipples.

Engorged milk duct becomes blocked and then infected.

*Staphylococcus* is the most common bacterial cause.

Mastitis is a breast infection without an abscess.

### Diagnosis

Clinical diagnosis with redness, tenderness, and warmth to a localized breast area

Abscess will often also have spontaneous drainage from nipple and palpable fluctuant mass.

Abscesses are frequently areolar or periareolar.

Bedside ultrasound can be used to demonstrate size and extent of breast mass.

## Management

For mastitis, patient should use warm or cold compresses, pain control, and continue breastfeeding.

Antibiotics should cover skin flora. Consider methicillin-resistant *Staphylococcus aureus* coverage as indicated.

For breast abscess, antibiotics and prompt surgical consultation

### 3. DELIVERY

Peggy Ochoa



**FIGURE 1.** Delivery with assistance from birth attendant. (From Klossner NJ. *Introductory Maternity Nursing*. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.)

#### Clinical Presentation

Known pregnant patient or unknown patient with rhythmic abdominal pain in every 2 to 3 minutes and lasting for 60 seconds

Pregnant patient complains of urge to “push” with pelvic, vaginal, or rectal pressure.

#### Diagnosis

Digital cervical exam confirms cervix is completely dilated.

Ultrasound confirms positive fetal heart tones.

Patient may have a broken bag of water or bloody mucous discharge.

The fetal head may be crowning when examining perineum.

#### Management

Have “birth on arrival” kit available (contains two cord clamps, scissors, bulb suction). Set up infant warmer with resuscitation equipment (Ambu bag with neonatal mask, suction, laryngoscope with 0/1 blade, and endotracheal tubes). Position mother on gynecology cart if available.

Hold towel or sponge between the vagina and anus and apply gentle pressure to support the perineum.

After head is delivered, instruct woman to stop pushing and check for nuchal cord (sweeping fingers around both sides of the neck). If nuchal cord is present, gently attempt to bring it over the head of the infant. If it cannot be loosened, double clamp it and cut between the clamps and then unwind.

Allow the head of the infant to restitute to one side or the other. Place both hands on either side of the head for support. Move hands downward with the head as the anterior shoulder delivers (do not apply traction to the neck—increased risk of shoulder dystocia).

Guide the body in an upward direction without pulling using a towel because the infant can be slippery.

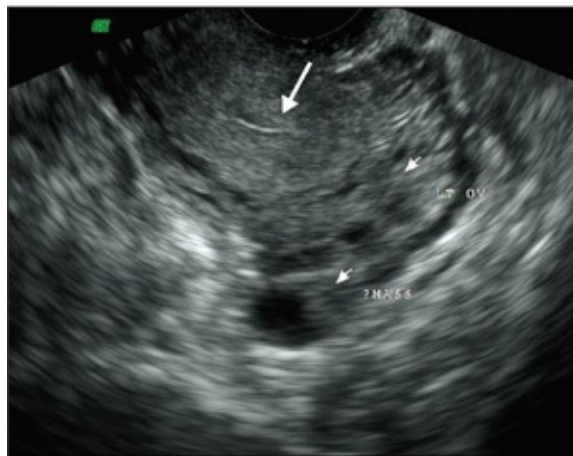
Double clamp and cut the cord. Suction newborns mouth, then nose, as needed.

## 4. ECTOPIC PREGNANCY

Dayle Whiteman-Davenport



**FIGURE 1.** Empty uterus with a decidual reaction resembling a gestational sac. (Courtesy of Tara Benjamin, MD, and Jill Wilson, MD, Indiana University School of Medicine, Department of Obstetrics and Gynecology.)



**FIGURE 2.** Transvaginal ultrasound image of left ectopic pregnancy. Uterus with thin lining of endometrium (*long arrow*) and left ovary with follicles and small mass (*smaller arrows*). (Courtesy of Kiwita Phillips, MD, and Franklyn Geary Jr., MD, Grady Memorial Hospital.)

### Clinical Presentation

Occurs in 1.5% to 2% of all pregnancies and accounts for 6% of all maternal deaths

Risk factors include pelvic inflammatory disease, previous tubal surgery, previous ectopic pregnancy, conception with an intrauterine device in place,

and assisted reproductive techniques (i.e., in vitro fertilization [IVF]). Patients with unruptured ectopic pregnancies present with bleeding and crampy abdominal or pelvic pain. Patients with ruptured ectopic pregnancies may present with peritonitis and/or signs of shock.

## Diagnosis

Urine pregnancy test in all females with vaginal bleeding and/or abdominal pain. If positive, obtain quantitative  $\beta$ -human chorionic gonadotropin (hCG) and transvaginal ultrasound. Draw blood for complete blood count, blood typing, and Rh determination.

With the exception of IVF-assisted pregnancy, if an intrauterine pregnancy (IUP) is identified, the frequency of coexisting ectopic pregnancy is 0.6 to 2.5:10,000.

At  $\beta$ -hCG levels above 1,500 to 3,000 mIU/mL, transvaginal ultrasound is 100% sensitive for diagnosing an IUP. If no IUP is seen, be suspicious for ectopic pregnancy.

Sonographic findings of an empty uterus with adnexal mass (other than simple cyst) are highly suggestive of ectopic pregnancy. Sonographic findings of empty uterus without an adnexal mass or free fluid are indeterminate, so correlate with serum hCG.

If serum hCG <1,500 mIU/mL, repeat quantitative hCG in 2 days. A normal IUP should show at least a 53% increase; ectopic pregnancy would show a slower rate of increase.

## Management

Unstable patients require two large-bore intravenous lines for rapid infusion of crystalloid and/or packed red blood cells to maintain blood pressure and immediate obstetrics/gynecology (OB/GYN) consultation.

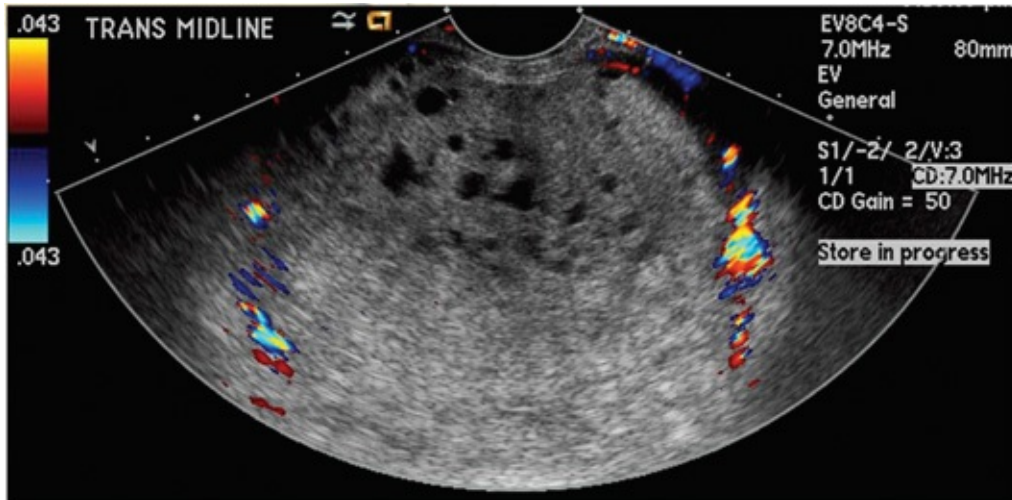
If the patient is stable, proceed with diagnostic workup. In reliable patients with indeterminate ultrasound results and hCG <1,000 mIU/mL, discharge with ectopic precautions and arrange follow-up in 2 days for repeat quantitative hCG, ultrasound, and OB/GYN reevaluation.

Definitive treatment should be determined by OB/GYN consultant and may involve laparoscopy, dilation and curettage, or medical management with methotrexate.



## 5. MOLAR PREGNANCY

Dayle Whiteman-Davenport



**FIGURE 1.** Molar pregnancy. (Courtesy of Kiwita Phillips, MD, and Franklyn Geary Jr., MD, Grady Memorial Hospital.)

### Clinical Presentation

Incidence is geographically dependent with 1 in 1,000 in the United States and 1 in 125 in Mexico and Southeast Asia.

Hydatidiform mole, or molar pregnancy, is the most common form of gestational trophoblastic disease.

Risk factors include extreme age of childbearing (i.e., <20 and >40 years) and history of molar pregnancy.

Most common presenting symptoms are vaginal bleeding and rapidly enlarging uterus. Other signs and symptoms include pelvic pain or pressure, anemia, hyperemesis gravidarum, hyperthyroidism, theca lutein cysts, and preeclampsia early in pregnancy (<20 weeks gestation).

The risk of malignant sequelae, including metastatic disease, is increased with complete hydatidiform moles and may be as high as 20%. Management may require a combination of chemotherapy, surgical, and medical therapy.

### Diagnosis

Majority have grossly elevated serum  $\beta$ -human chorionic gonadotropin ( $\beta$ -

hCG) level for gestational age, so serum  $\beta$ -hCG and pelvic ultrasound are the initial diagnostic step.

Ultrasound findings vary from the classical image of a “snowstorm” to the complex mass of swollen hydropic trophoblastic tissue seen as a “cluster of grapes.” Large theca lutein cysts are common and may lead to ovarian torsion. Complete blood count; coagulation studies; and liver, renal, and thyroid function status should be assessed as well as blood type and antibody screen. Chest x-ray to rule out metastatic disease *prior* to evacuation is recommended because the trophoblast can embolize during evacuation and lead to an assumption of metastasis.

## Management

Suction curettage is the optimal method of evacuation in patients who wish to retain reproductive function.

Hysterectomy is an acceptable option for a woman who has completed her childbearing years because it is associated with a lower risk of local invasion, although it does not eliminate metastatic disease.

All uterine contents should be sent to pathology for histologic diagnosis with flow cytometry to determine karyotype.

Despite evacuation of uterine contents, the patient remains at risk for invasive trophoblastic disease or choriocarcinoma. Serial serum  $\beta$ -hCG should be performed until undetectable. Patients should be placed on birth control (barrier methods or oral contraceptives) throughout the surveillance period.

## 6. UTERINE PROLAPSE

Mark P. Kling



FIGURE 1. Stage 4 uterine prolapse.

### Clinical Presentation

Risk factors are multiparity, obesity, age, and chronic constipation.  
Complaint of vaginal bulge or something “falling out” of the vagina  
Associated symptoms: stress incontinence, constipation, and sexual dysfunction  
Associated findings: urinary obstruction and hydronephrosis

### Diagnosis

Diagnosis is based on examination.  
Perform the pelvic exam in dorsal lithotomy position but also while the patient strains or stands to test for prolapse.

## Management

Gynecologic consultation or referral, typically nonemergent

Conservative approach includes vaginal pessaries, vaginal pessaries and pelvic floor exercises.

Surgery may be performed in patients who fail or decline conservative management.



SECTION

K

DERMATOLOGIC

SECTION EDITOR  
Erik Nordquist

# 1. ANIMAL BITES

John Hardwick, Joseph Weber



FIGURE 1. Cat bite.



FIGURE 2. Dog bite.

## Clinical Presentation

Seventy-five percent of animal bites are inflicted by a pet owned by the victim.

Canines account for the overwhelming majority of animal bites.

Around 15% of cat and dog bites become infected. Dog bite infections are most commonly purulent, non-abscess-forming lesions. Cat bite infections most commonly present as cellulitis with or without lymphangitis.

Cat and dog bite wound infections are generally polymicrobial with both



aerobic and anaerobic organisms. *Pasteurella*, *Streptococcus*, *Staphylococcus*, and *Fusobacterium* are the most common isolates.

Cats are the main reservoir for *Bartonella henselae*, the causative organism of cat scratch fever. Typical symptoms consisting of focal lymphadenopathy and fever arise around 1 to 3 weeks after inoculation via cat scratch or bite.

## Diagnosis

High-risk features of mammalian bites for developing infection or resulting in poor cosmesis include wounds that penetrate bone, joint, or tendon; wounds that present >8 hours from injury; puncture wounds or crush wounds; wounds that occur in the immunocompromised; and wounds on the hands, face, or genitals.

Consider radiographs in any bite wound directly overlying bone or joint.

## Management

Thoroughly irrigate the wound and debride any devitalized tissue.

Bite wounds to the hand should be left open because they are at an increased risk of infection. Wounds elsewhere may undergo primary closure if they are considered low risk for infection.

Prophylactic antibiotics are controversial but should be started in any patient at high risk for infection.

Seven to 10-day course of amoxicillin-clavulanate is treatment of choice; doxycycline/clindamycin for penicillin allergy; doxycycline alone if cat scratch fever is suspected

Tetanus vaccination is necessary if it has been >5 years since last tetanus booster.

In the United States, rabies is incredibly rare among rodents, dogs, and cats; therefore, vaccination is generally not indicated. Consider infectious disease consultation and rabies vaccination in any bite sustained from high-risk animals including bats, foxes, coyotes, raccoons, and skunks.



## 2. ATOPIC DERMATITIS

Eli Poorvu, Mike Schindlbeck



FIGURE 1. Atopic dermatitis of the digits.



FIGURE 2. Severe atopic dermatitis of the hand.

### Clinical Presentation

The underlying cause of atopic dermatitis, otherwise known as *eczema*, is not clearly understood.

The overwhelming majority of patients will experience symptoms by the age of 5 years.

The prevalence of atopic dermatitis in the United States is approximately 12%.

### Diagnosis

The clinical manifestations tend to vary by the age of the patient, although nearly all suffer from dry skin and pruritus.

The infantile stage occurs in patients younger than 2 years of age and is characterized by erythematous, scaly, crusted, and sometimes exudative patches over the cheeks and extensor surfaces of extremities.

The childhood stage occurs in patients between 2 and 12 years of age and is characterized by skin thickening and excoriation secondary to severe pruritus. The flexor surfaces of the extremities and volar surfaces of the wrists, ankles, and neck are most commonly affected.

The adult stage occurs after the age of 12 years. Symptoms tend to be more localized, characterized primarily by lichenified lesions over the flexor surfaces and occasionally the face.

## Management

The management of atopic dermatitis focuses primarily on symptom control and prevention.

Topical moisturizing solutions can be used to treat dry skin, although varieties with high water contents should be avoided because they may exacerbate the condition.

Antihistamines, such as diphenhydramine, can be used to help alleviate the associated pruritus.

Low-dose topical corticosteroids should be considered first-line therapy for active symptoms, although their use on the face should be limited due to the potential for secondary thinning or atrophy of the skin.

Topical calcineurin inhibitors (e.g., tacrolimus or pimecrolimus) can be used in patients older than the age of 2 years refractive to topical steroids, although long-term use should be avoided due to potential neoplastic concerns.

Severe symptoms refractory to topical treatments may require localized phototherapy or more aggressive systemic therapy. Consider dermatology referral or consultation in these patients.

### 3. AUTOINJECTOR INJURY

Joseph Palter, Rashid Kysia



**FIGURE 1.** Left thumb puncture wound and thumb pad pallor.  
(Reprinted from Sherman SC. Digital Epipen® injection: a case of conservative management. *J Emerg Med.* 2011;41(6):672–674. Copyright 2011, with permission from Elsevier.)



**FIGURE 2.** Left thumb nail bed with minimal capillary perfusion.  
(Reprinted from Sherman SC. Digital Epipen® injection: a case of conservative management. *J Emerg Med.* 2011;41(6):672–674. Copyright 2011, with permission from Elsevier.)

## Clinical Presentation

Incidence of 1 per 50,000 epinephrine autoinjectors issued  
Patients will typically endorse recent handling of autoinjector device with accidental discharge into fingers.

## Diagnosis

Reported history of accidental injection is key to diagnosis because one may not be able to visualize the actual puncture wound.

Skin pallor of injected finger with prolonged or absent capillary refill

Affected finger cool to touch when compared to others

Loss of pain and temperature sensation, loss of all sensation, subjective paresthesia and/or pain

## Management

Conservative management: warm compresses or soaks, digital massage, application of nitroglycerine paste

Complete resolution of symptoms with conservative approach range from 6 hours to 10 weeks but most often within hours to a few days.

Invasive management: local injection of phentolamine (0.5 to 3.5 mg) *or* terbutaline (0.1 to 0.3 mg) either of which can be mixed with lidocaine (1 mL of 1% to 2% solution)

Resolution of symptoms with invasive approach is typically <5 to 30 minutes.

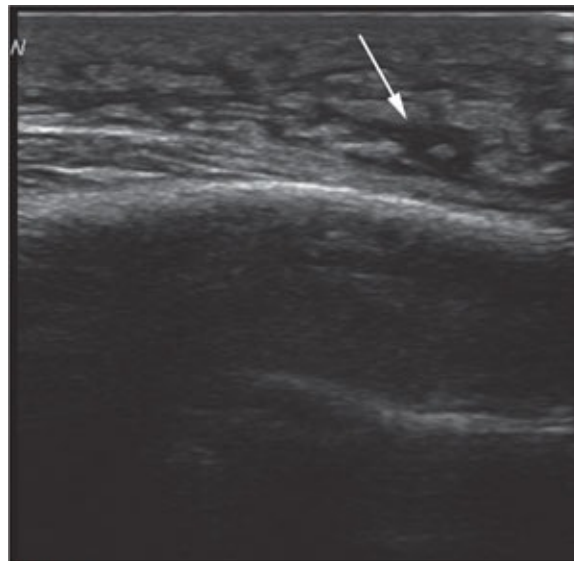
Regardless of treatment type, there are no reported cases of digital necrosis resulting from autoinjector injuries.

## 4. CELLULITIS

Erin Clark, Steven Aks



**FIGURE 1.** Cellulitis following surgery.



**FIGURE 2.** Ultrasound image demonstrating cobblestone edema (arrow) consistent with cellulitis.

### Clinical Presentation

Cellulitis is a soft tissue infection of the skin and subcutaneous tissue. Patients typically present with localized tenderness, pain, swelling, warmth, and blanching erythema that spreads if left untreated.

Most common location is the lower extremities.

*Staphylococcus aureus* and *Streptococcus pyogenes* are the most common organisms.

Risk factors include obesity, venous insufficiency, trauma, and lymphedema.

## Diagnosis

Diagnosis is based on history and physical exam.

Fever is uncommon and suggests a more complicated infection. Vital signs are typically normal.

White blood cell count is usually normal or mildly elevated with little to no left shift.

Ultrasonography may help differentiate between cellulitis and abscess.

## Management

Antibiotics should be directed at skin flora.

An oral outpatient regimen may be appropriate in immunocompetent, well-appearing patients.

Patients who are immunocompromised, who have signs or symptoms or systemic infection, or who have hand/foot involvement may require intravenous antibiotics.

Consider methicillin-resistant *S. aureus* in patients with an associated abscess or in those not responding to antibiotics.



## 5. COLD PANNICULITIS

Jessica Folk



FIGURE 1. Infant with cold panniculitis to bilateral cheeks.

### Clinical Presentation

Nodular, erythematous rash on areas exposed to cold

Inflammation of subcutaneous adipose tissue

Typically affecting cheeks and foreheads in infants/children

Obese adult females develop nodules on the buttocks, thighs, arms, or under the chin.

### Diagnosis

History providing a cold exposure (e.g., ice therapy, eating a Popsicle)

Differential: frostbite, lupus erythematosus, scleroderma, vascular malformation

Punch biopsy is typically inadequate as findings are localized to subcutaneous tissue.

## Management

Self-limiting

Symptomatic relief

Slow rewarming

## 6. CONTACT DERMATITIS

Eli Poorvu, Mike Schindlbeck



**FIGURE 1.** Contact dermatitis after exposure to poison ivy. Note the erythematous, weeping skin with vesicle formation.

### Clinical Presentation

Contact dermatitis refers to a localized irritation of the skin induced by direct exposure to a particular substance.

Cases can be divided clinically into irritant and allergic subtypes.

Allergic contact dermatitis represents a T cell–mediated immune response secondary to an allergen exposure. Patients typically present with pruritus and erythematous plaques that may develop into vesicles or bullae, and chronic exposures may lead to dry fissured skin and lichenification.

Irritant contact dermatitis is caused by direct skin exposure to a particular irritating substance and presents with pruritus, erythema, dry scaling skin, and fissuring.

### Diagnosis

Diagnosis is based on history and physical exam.

Exposure to plants of the *Toxicodendron* genus (poison ivy, oak, and sumac) is the leading cause of allergic contact dermatitis in North America. Additional etiologies include exposure to certain metals (nickel, gold), soaps and laundry

detergents, and topical medications.

Common causes of irritant contact dermatitis include detergents and surfactants, strong acids or alkalis, and physical irritants such as fiberglass. A linear distribution of lesions is highly consistent with a plant exposure.

## Management

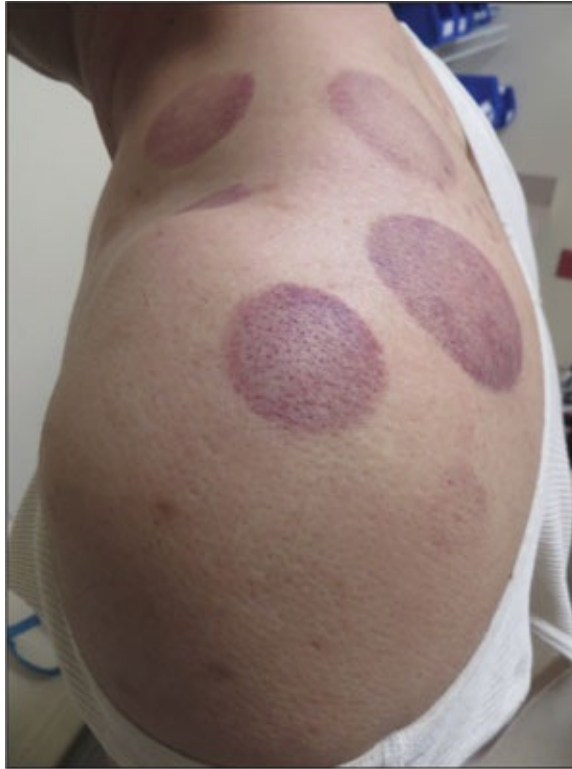
Removal of the causative agent and prevention of further exposure is the first and most important step in treatment.

Allergic contact dermatitis should be treated with topical corticosteroids and oral antihistamines. Calamine lotion, oatmeal baths, and cool wet compresses may be helpful for symptomatic relief. A slow taper of systemic steroids may be necessary in severe cases, such as those that involve the face or genitals.

Irritant contact dermatitis should be treated with topical corticosteroids and emollients to restore normal skin integrity.

## 7. CUPPING/SPOONING

Ernesto J. Romo, Steven H. Bowman



**FIGURE 1.** Cupping to the back, shoulder, and neck.



**FIGURE 2.** Cupping on the back.

## Clinical Presentation

Both are common traditional medicine therapies used in parts of Asia, Eastern Europe, Latin America, and Middle East.

Patients typically present for evaluation of another complaint or symptom, such as abdominal pain or fever and chills.

In cupping, a small glass, ceramic, or metal vessel is placed on the body and heated, creating a vacuum and the resulting well-defined, circular lesions.

Spooning is performed by firmly rubbing oiled skin with a spoon.

## Diagnosis

Diagnosis is based on history and physical exam.

Cupping and spooning are generally incidental findings. Investigate the patient's primary concern.

Consider other possible etiologies, including abuse, burn, coagulation disorder, or vasculitis.

## Management

If clinical suspicion of abuse, report to proper authorities.

Treat appropriately for burn if present.

Symptomatic care for pain

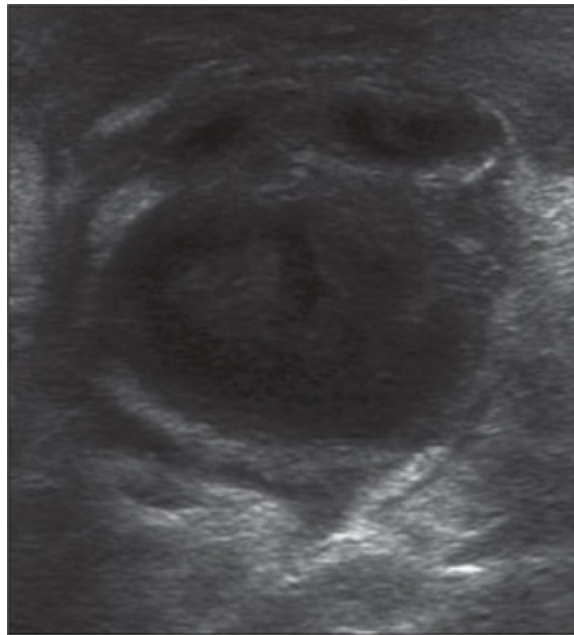


## 8. CUTANEOUS ABSCESS

Erin Clark, Steven Aks



**FIGURE 1.** Abscess formation in an IV drug abuser.



**FIGURE 2.** Ultrasound image of an abscess.

### Clinical Presentation

An abscess is a localized collection of purulent fluid that causes a painful, fluctuant, soft tissue swelling.

May occur in any area of the body

The bacteria in the abscess typically reflects the normal skin flora of the involved body part.

## Diagnosis

Diagnosis is based on history and physical exam.

The patient will complain of a painful, enlarging soft tissue mass.

Fever and systemic symptoms suggest more extensive tissue involvement or bacteremia.

Ultrasonography may help localize deeper abscesses as well as differentiate abscesses from cellulitis.

## Management

Incision and drainage is the definitive treatment. The incision should be large and deep enough to allow for adequate drainage. Loculations should be decompressed.

Antibiotics are not indicated in patients who are immunocompetent. In those patients without normal host defenses, such as diabetics or patients with HIV, antibiotics are indicated.

Selection of antibiotics should cover skin flora, including methicillin-resistant *Staphylococcus aureus*, if suspected.

## 9. CUTANEOUS LARVA MIGRANS

Audrey Herbert, Deborah Kimball



**FIGURE 1.** Cutaneous larva migrans. The skin shows a creeping eruption with the characteristic of serpiginous, raised lesion. (From Farrar WE, Wood MJ, Innes JA, et al. *Infectious Diseases: Text and Color Atlas*. 2nd ed. New York, NY: Gower Medical Publishing; 1992.)



**FIGURE 2.** Cutaneous larva migrans on the foot. (From Goodheart HP. *Goodheart's Photoguide of Common Skin Disorders*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.)

### Clinical Presentation

Skin condition caused by a hookworm parasite that infects the intestines of dogs, cats, and wild animals as primary hosts

Hookworm larva come into direct contact with human skin in areas contaminated with animal feces, most commonly beaches of Asia, Africa, and South America.

The hookworm larva penetrates and causes red, serpiginous, dramatically pruritic lesions that arise 1 to 5 days after exposure.

## Diagnosis

Diagnosis is usually made clinically based on the appearance of lesions, severe pruritus, and a history of travel to potentially contaminated areas. Secondary bacterial infections may arise due to excoriation, potentially making identification of the initial lesion difficult.

## Management

Albendazole is the treatment of choice. If untreated, the larva will usually die within weeks to months, as they are unable to penetrate the basement membrane in the incidental host. Treatment of secondary bacterial infections with topical or oral antibiotics may be necessary.

## 10. DRUG ERUPTIONS

Elisabeth Giblin, Michele Zell Kanter



FIGURE 1. Drug eruption due to a cephalosporin antibiotic.

### Clinical Presentation

Skin appearance can be varied and include papulosquamous lesions, urticaria, pustules, and bullous lesions.

Hypersensitivity reactions typically occur within 8 weeks of initiating drug therapy.

Frequently caused by antibiotics, anticonvulsants, barbiturates, nonsteroidal antiinflammatory drugs, or phenothiazines; however, all medications should be considered.

### Diagnosis

Examine airway for signs of respiratory compromise, angioedema, or tongue swelling.

Examine the total body surface area with attention to mucous membranes

(conjunctiva, genitourinary, oral mucosa).

Laboratory tests should include complete blood count with differential, serum chemistry studies, liver function tests, and urinalysis.

## Management

The goal is to identify and discontinue the causative agent.

Treatment is supportive and symptom-based: antihistamines, corticosteroids, and topical antihistamines.

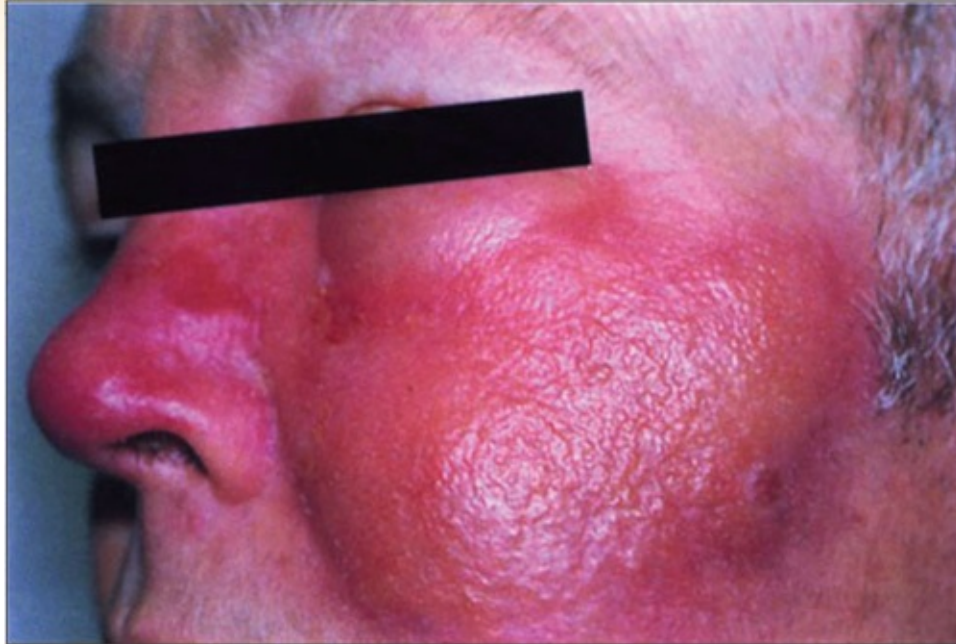
May take 2 to 4 weeks for eruption to diminish after discontinuation of offending agent

Educate patient to avoid the offending agent to prevent reexposure.



## 11. ERYSIPELAS

Isam Nasr



**FIGURE 1.** Erysipelas. A 51-year-old man had fever, chills, and an expanding sharply marginated erythematous edematous plaque on the cheek. (From Elder DE, Elenitsas R, Rubin I, et al. *Atlas and Synopsis of Lever's Histopathology of the Skin*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

A bacterial infection involving the upper dermis that characteristically extends into the superficial cutaneous lymphatics

Eighty-five percent of cases occur on the legs and are caused by *Streptococcus pyogenes*, but other bacteria including nongroup A *Streptococcus*, *Staphylococcus*, and even gram-negative species can be responsible.

Erysipelas begins as a small erythematous patch that progresses to a fiery-red, indurated, tense, and shiny plaque.

The lesion classically exhibits raised, sharply demarcated, advancing margins. Complications include abscess formation, gangrene, and thrombophlebitis.

### Diagnosis

Diagnosis is based on history and physical exam.

Differential diagnoses include cellulitis, contact dermatitis, erysipelas



carcinomatosum, erythema induratum, granuloma faciale, stasis dermatitis, and systemic lupus erythematosus.

Laboratory workup is not required for the diagnosis or treatment.

## Management

Elevation and rest of the affected limb are recommended to reduce local swelling, inflammation, and pain.

Penicillin is the first-line agent for the treatment of moderately severe infections, whereas nafcillin is reserved for severe infections and for penicillin-resistant streptococcal or staphylococcal infections.

A first-generation cephalosporin or macrolide may be used if the patient has an allergy to penicillin.

## 12. ERYTHEMA MULTIFORME

Jason Lee, Mark P. Kling



**FIGURE 1.** Erythema multiforme in a child. (From Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)



**FIGURE 2.** Raised target lesions of erythema multiforme.

## Clinical Presentation

A hypersensitivity reaction usually occurring in response to a medication or more commonly an infection (e.g., herpes simplex virus and mycoplasma). Skin lesions are raised target or iris lesions distributed symmetrically over the extremities and trunk with concentric zones of color change. Palm and sole involvement is common.

May involve one area of mucosa, the extent of which is variable. Most mucosal lesions occur on the mouth; ocular involvement can also occur.

## Diagnosis

Based on history and clinical findings

Referral for histopathologic testing may be required to confirm the diagnosis and help differentiate it from other rashes with similar appearance.

## Management

Treat the precipitating infection or remove of the causative agent.

Further treatment is tailored to the patient based on the severity of the disease and the extent of mucosal involvement.

Mild cutaneous involvement is managed with achieving symptomatic improvement.

Extensive mucosal involvement resulting in poor oral intake may require fluid and electrolyte management; ocular involvement warrants ophthalmology consultation.

## 13. ERYTHEMA NODOSUM

P. Quincy Moore, Shari Schabowski



**FIGURE 1.** Erythematous subcutaneous nodules on the anterior surfaces of the legs.

### Clinical Presentation

Most common type of panniculitis. Six times more common in women than men

Symmetrical, painful, erythematous subcutaneous nodules on the anterior surfaces of both legs; can also appear on the thighs, trunk, and extensor surfaces of the upper extremities but pretibial nodules are almost always present

Polyarthralgia, fever, and malaise may accompany or precede skin findings. Most commonly seen with streptococcal pharyngitis, especially in children, but may be seen in association with inflammatory bowel disease, cancer, sarcoidosis, bacterial or fungal infections, tuberculosis, antibiotic use, or oral

contraceptive pills

## Diagnosis

Diagnosis is based on history and physical exam.

May be a clue in the diagnosis of a systemic medical problem; therefore, basic workup includes complete blood count, basic metabolic panel, liver function tests, antistreptolysin-O titer, and chest x-ray. Biopsy is usually not required. Erythrocyte sedimentation rate is elevated in 15% to 40% of cases.

## Management

Typically resolves without intervention or scarring in 2 to 8 weeks

Treatment is symptomatic, often starting with nonsteroidal antiinflammatory drugs. Treatment of the underlying disorder, if identified, is also important. Antibiotics or glucocorticoids are generally not indicated.

## 14. HERPES ZOSTER

Lisa R. Palivos



**FIGURE 1.** Multiple vesicles on an erythematous base in a dermatomal distribution.

### Clinical Presentation

Caused by reactivation of latent varicella-zoster virus

Prodrome of pain and paresthesias develop initially in a dermatomal distribution.

This is followed by the classic rash (clusters of vesicles on an erythematous base), which is usually unilateral and limited to one to two dermatomes.

The most common dermatomes are thoracic, lumbosacral, and trigeminal.

Ramsay Hunt syndrome involves cranial nerve VII palsy with vesicles in auditory canal and auricle.

Hutchinson's sign (lesions at the tip of the nose) can signal eye involvement.

### Diagnosis



Diagnosis is based on history and physical exam.

## Management

Oral antiviral therapy (acyclovir, famciclovir, or valacyclovir) within 72 hours of eruption for immunocompetent patients

Intravenous acyclovir for immunocompromised patients

Pain medication and consider referral to pain specialist for nerve blocks.

Ophthalmology consult for eye involvement

## 15. HIDRADENITIS SUPPURATIVA

P. Quincy Moore, Shari Schabowski



**FIGURE 1.** Draining nodules and scarring in the axilla in a patient with hidradenitis suppurativa. (From Goodheart HP. *Goodheart's Same-Site Differential Diagnosis: A Rapid Method of Diagnosing and Treating Common Skin Disorders*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Usually occurs between puberty and age 40 years

Recurrent, painful, draining nodules typically found in intertriginous skin areas

Usually begins as inflammatory, pimple-like nodules that occur in areas where pimples are not typical—axilla, groin, perianal, perineal, and inframammary regions

Nodules may regress spontaneously in one to several weeks or progress to open comedones or abscesses. Deep scarring and interconnecting sinus tracts may be present.

### Diagnosis

Diagnosis is based on characteristic lesions, location, and chronic relapsing course.

Not an infection, so bacterial cultures and immunodeficiency workup are not

indicated

## Management

Clindamycin 1% lotion twice daily

Antibacterial soaps such as chlorhexidine once to twice weekly

Incision and drainage is rarely helpful and can increase scarring.

Referral to a specialist should be considered for additional treatment options.

## 16. IMPETIGO

Aaron Ginster, Mark P. Kling



FIGURE 1. Impetigo of the face.

### Clinical Presentation

Isolated skin infection with no systemic symptoms, such as fever

Most often caused by group A  $\beta$ -hemolytic *Streptococcus* or *Staphylococcus aureus*

Presents as rash occurring most frequently on the face, neck, and extremities

Occurs most commonly in 2- to 5-year-olds

There are two forms: nonbullous impetigo and bullous impetigo. The nonbullous form is most common and consists of initial macules and papules that develop into thin-walled vesicles. These eventually rupture, leaving a yellow serous drainage that hardens and forms the characteristic appearance of honey-colored crust. The bullous form is part of the staphylococcal scalded skin syndrome spectrum and is toxin-mediated. Instead of forming vesicles, bullae are formed (usually <3 cm), which are most often on extremities.

Both forms usually resolve spontaneously in 2 weeks.

## Diagnosis

Diagnosis is based on history and physical exam.

Culture may be used when patients fail to respond to standard treatment, or methicillin-resistant *S. aureus* (MRSA) is suspected.

## Management

Good hygiene, daily cleansing, and discouragement of scratching that leads to spread

Topical antibiotics, most commonly mupirocin, are used for both forms.

Use oral antibiotics when large areas are affected or when topical application is not possible.

Cephalexin, amoxicillin–clavulanate, or dicloxacillin may be used for oral treatment.

If MRSA is suspected, doxycycline or trimethoprim-sulfamethoxazole may be used.

## 17. INTERTIGO

Erin Clark, Steven Aks



FIGURE 1. Intertrigo of the groin.

### Clinical Presentation

Intertrigo is a dermatitis that results from irritation of skin folds due to friction, moisture, and warmth.

Most commonly occurs in groin and inframammary folds

Appears as erythematous, macerated fissures in skin fold areas

Areas may be pruritic and/or burning.

### Diagnosis

Diagnosis of exclusion

Streptococcal and candidal infections, as well as allergic dermatitis, may appear similarly.

Satellite lesions may be present in the above conditions but are not typical of intertrigo.

## Management

Keep affected areas dry and cool to promote resolution; avoid potential irritants.

Zinc oxide paste to protect against moisture

Treat secondary bacterial or fungal infections.



## 18. JAUNDICE

Theresa Kim, Rebecca Roberts

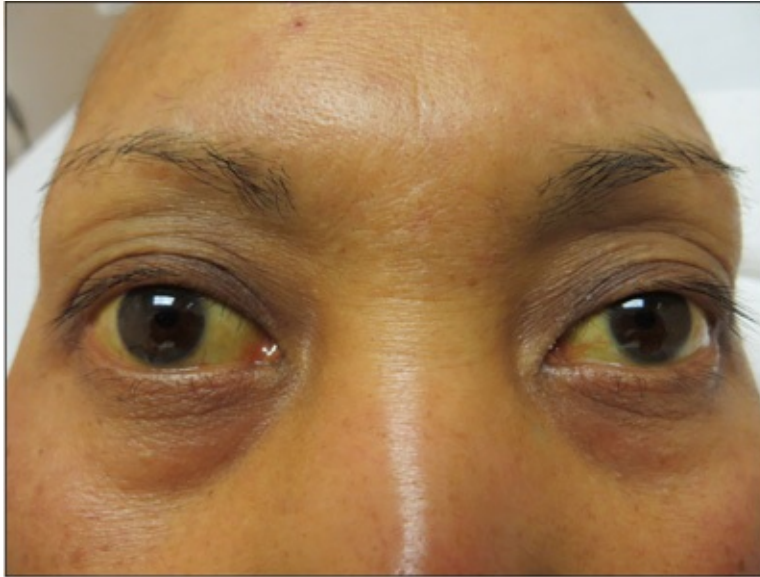


FIGURE 1. Jaundice of the skin with scleral icterus.

### Clinical Presentation

Seen best in natural lighting, jaundice is a yellow discoloration of the sclera, skin, and mucous membranes caused by hyperbilirubinemia.

Two types of hyperbilirubinemia exist: unconjugated and conjugated.

Unconjugated hyperbilirubinemia is caused by increased bilirubin production usually due to hemolysis or impaired ability of the liver to uptake or conjugate bilirubin. Conjugated hyperbilirubinemia is caused by intra- or extrahepatic cholestasis or decreased excretion of conjugated bilirubin by the gastrointestinal system.

### Diagnosis

On history, ask about pruritis, pale-colored stools, dark tea-colored urine, abdominal pain, weight loss, anorexia, fevers, malaise, alcohol abuse, prior abdominal surgeries, or family history of jaundice.

Physical exam may reveal jaundice, ascites, spider telangiectasis, palmar erythema, organomegaly, and right upper quadrant abdominal pain.

Elevated serum bilirubin, liver enzymes, coagulation profile, acute and chronic hepatitis panels, serum and urine drug screens, and bilirubinuria on urinalysis help to differentiate the causes of jaundice.

Ultrasound is the preferred initial imaging technique but also consider computed tomography of the abdomen and pelvis.

## Management

Treatment modalities differ based on the cause of jaundice and range from supportive to parenteral antibiotics to surgery.

Patients with new-onset jaundice and appropriate follow-up may potentially be discharged from the emergency department if there is no evidence of obstruction, infection, hemodynamic instability, or liver failure.

Examples of cases requiring specific immediate interventions are (1) infants with markedly elevated bilirubin  $>20$  mg/dL or severe anemia in the setting of autoimmune hemolytic disease and (2) patients with extrahepatic biliary obstruction necessitating emergent surgical and/or gastrointestinal consultation.

## 19. LEVAMISOLE VASCULITIS

Neera Khattar



**FIGURE 1.** Diffuse purpura of the leg in a net-like formation in a patient with cocaine abuse.

### Clinical Presentation

New-onset vasculitis in a patient with confirmed or suspected cocaine intoxication

Cutaneous lesions are typically retiform purpura, most commonly visualized on the ears but can also be found on face, extremities, or diffusely.

Hemorrhagic bullae with or without necrosis are less common but may accompany purpura.

Cutaneous presentation is often associated with arthralgia; ear, nose, and throat complaints; and generalized malaise.

Levamisole, a veterinary anthelmintic agent, is a common adulterant of cocaine.

### Diagnosis

Presence of purpura and cocaine on urine toxicology screen is initially diagnostic.

Skin biopsy of purpura can confirm diagnosis.

Diagnosis may be supported by a positive urine levamisole screen, but a negative urine screen cannot be used to rule out levamisole toxicity. Positive lab findings of neutropenia and polyspecific antineutrophil cytoplasmic antibodies confirm levamisole toxicity.

## Management

Purpura is typically self-resolving with cessation of cocaine use.

If no necrosis present, give supportive treatment with pain control, intravenous fluids, and management of cocaine intoxication and withdrawal.

If cutaneous lesions involve necrosis, surgical debridement is necessary followed by wound care and monitoring for wound infection.

## 20. LICE

Thomas W. Engel II



**FIGURE 1.** Adult body louse.



**FIGURE 2.** Nits (larvae) of head lice. (Reproduced with permission from Goodheart HP. *Goodheart's Photoguide of Common Skin Disorders*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.)

### Clinical Presentation

Seen mostly in school-aged, light-skinned children  
Commonly transmitted between close household contacts  
Found on the scalp, mostly involving the occipital or posterior auricular area but also in other skin folds and the pubic region

Asymptomatic for up to 6 weeks while the female parasites lay eggs, known as *nits*, and feed off of human blood multiple times per day

When patients become symptomatic, they will have intense pruritus with erythematous macules or wheals, which can become secondarily infected with scratching.

## Diagnosis

White dots near the base of hair follicles are presumptive of nits and often are the only sign of infection.

Blue or black grains indicative of adult lice are rarely seen.

A magnifying glass and fine comb add in the detection of nits.

A microscopic exam of hair from the affected region is definitive but generally not necessary.

## Management

Permethrin and malathion are first-line. Repeat treatment in 7 days with daily fine comb nit removal.

Treatment failure from improper application or rapid reinfestation; however, ivermectin and lindane can also be used in select patients.

All close contacts require screening; sexual partners require reflex treatment. Clothing, hair items, and bedding all require either laundering, boiling, or to be sealed in a plastic bag for 2 weeks. Fumigation is not necessary.



## 21. LYME DISEASE

Trevor Lewis



FIGURE 1. Erythema migrans.

### Clinical Presentation

Lyme disease is a tick-borne illness caused by the bacterium *Borrelia burgdorferi*. Transmission occurs via an infected tick bite.

Symptoms include fever, headache, fatigue, muscle aches, tender adenopathy, and a characteristic rash called *erythema migrans*.

Late disease symptoms include joint arthritis and effusion, temporary paralysis of facial muscles (Bell palsy), and severe fatigue.

### Diagnosis

History can reveal possible exposure or travel to endemic areas.

Rash of erythema migrans is present in 70% to 80% of infected patients and usually occurs at the initial bite site. Average time from inoculation is 7 days (range 3 to 32 days). As rash expands, the center may clear leading to a “bulls-eye” appearance.

Enzyme-linked immunosorbent assay (ELISA) blood test measures antibodies



against the Lyme bacteria. The test may not become positive until 2 weeks after infection.

## Management

Standard treatment for Lyme disease is a 14- to 28-day course of antibiotics. Common regimens include doxycycline, amoxicillin, or cefuroxime.

Treatment should begin immediately based on identification of erythema migrans rash because blood testing is not reliable early in the course of infection.

ELISA test only indicates prior infection. Treatment should be based on signs and symptoms coupled with positive test.

## 22. MENINGOCOCCEMIA

Sean Dyer, Mark Mycyk



**FIGURE 1.** Young boy with meningococcal infection. (From Fleisher GR, Ludwig W, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)



**FIGURE 2.** Characteristic purpuric and petechial rash of meningococcemia. (From Betts RF, Chapman SW, Penn RL. *Reese and Betts' A Practical Approach to Infectious Diseases*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2002.)

### Clinical Presentation

Early symptoms may be mild and nonspecific: fever, headache, myalgias, vomiting, and malaise.

The characteristic petechial or purpuric nonblanching rash may be absent early in the course of illness.

Sudden deterioration and rapid progression to death is common without treatment.

### Diagnosis

Suspect the diagnosis in patients with a fever, a characteristic rash, and severe illness.

Abnormal serum tests include leukocytosis, coagulopathy, and acidemia.

Definitive diagnosis is confirmed with cerebrospinal fluid analysis (Gram stain, cell count, culture) or blood culture.

Do not allow diagnostic workup to postpone resuscitation or administration of antibiotics in suspected cases.

## Management

High-dose penicillin or third-generation cephalosporin should be administered early.

Early corticosteroid therapy may prevent neurologic injuries and improve adrenal gland response in severe cases.

Remember prophylactic antibiotics for close contacts within the first 24 hours.

Ensure droplet precautions during evaluation and treatment.

## 23. NECROTIZING FASCIITIS

Rosaura Fernandez



**FIGURE 1.** Necrotizing soft tissue infection on the buttock.



**FIGURE 2.** CT image in the same patient demonstrating gas formation.

### Clinical Presentation

Direct invasion of bacteria through a break in the skin leading to a necrotizing soft tissue infection of subcutaneous fat, fascia, or muscle

Rare condition with a rapid spread of infection and high mortality of 20% to 80%

Signs of fever and tachycardia are typical but can advance to sepsis or septic

shock rapidly.

Risk factors include diabetes, intravenous (IV) drug or alcohol abuse, obesity, immune compromise, and peripheral vascular disease.

## Diagnosis

Must have a high clinical suspicion for this condition with any presenting soft tissue infection

Diagnosis is made by surgical exploration and direct visualization of underlying soft tissue and fascia.

Affected areas may have marked erythema, edema, and intense pain out of proportion to exam findings.

Advanced skin changes can demonstrate a deep red or purple color to a dusky grey color, formation of bullae, reduced sensation of involved area, crepitus, and signs of compartment syndrome.

Plain radiographs and computed tomography may detect subcutaneous air, although an absence of air does not rule out the condition, and obtaining imaging should not delay surgical consultation or treatment.

## Management

Treat with the aggressive administration of IV fluids and vasopressors, if needed.

Initiate broad-spectrum antibiotic coverage against gram-positive, gram-negative, and anaerobic bacteria.

Definitive management is surgical exploration and debridement with possible fasciotomy and amputation.

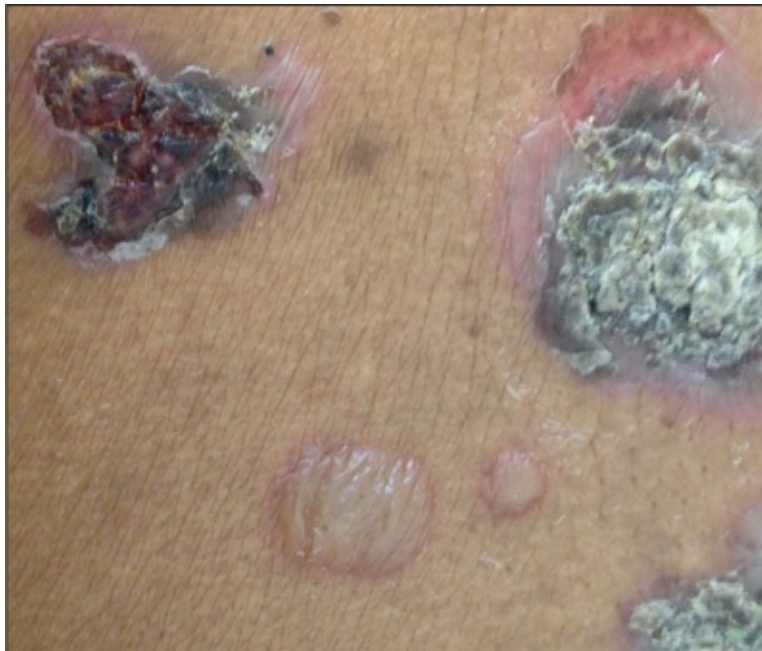
Prognosis is dependent on the site involved, extent of infection, patient characteristics, and time to treatment.

## 24. PEMPHIGUS VULGARIS

Eli Poorvu, Mike Schindlbeck



**FIGURE 1.** Pemphigus vulgaris on the back.



**FIGURE 2.** A small bullae with several partially healing lesions.



## Clinical Presentation

Pemphigus vulgaris is an autoimmune disorder characterized by blistering lesions of the skin and mucosal surfaces.

The incidence is rare, with approximately 0.5 cases per 100,000 individuals. Mortality rates are reported between 5% and 15% with appropriate treatment. Lesions on the skin appear as flaccid bullae filled with a clear fluid, whereas mucosal lesions manifest as painful erosions, with the oral mucosa most commonly affected.

## Diagnosis

The Nikolsky's sign (blister formation when the skin is rubbed at the edge of an existing lesion or over normal skin) is typically present in pemphigus vulgaris and can be used to distinguish it from other blister-forming disorders such as bullous pemphigoid.

Skin biopsy can be used to make the definitive diagnosis.

## Management

Oral corticosteroids are the mainstay of therapy and generally produce a rapid improvement in symptoms, although there is currently no definitive evidence to support an optimum regimen.

Immunosuppressive agents should be considered early in the course of the disease.

Local wound care with blister debridement and antibiotic use for secondary infection is essential. Oral lesions can be treated with triamcinolone paste to improve healing and viscous lidocaine or other anesthetics for symptomatic relief.



## 25. PILONIDAL CYST

P. Quincy Moore, Shari Schabowski



FIGURE 1. Pilonidal cyst.

### Clinical Presentation

Most frequently seen in males in their late teens and early 20s

Risk factors include sedentary lifestyle, obesity, family history, local trauma, and deep intergluteal cleft.

### Diagnosis

Presentation is variable and can include an asymptomatic pilonidal cavity or sinus, a pilonidal abscess, or a chronic inflammation and drainage.

Symptomatic presentations usually include pain while sitting or performing activities that stretch the affected area. Patients may report purulent and/or sanguineous drainage.

Clinical diagnosis is based on history and exam findings of a pore, sinus, or abscess located in the midline at the apex of the superior gluteal fold. A tender, swollen, and fluctuant nodule should raise concern for an abscess.

Fever and malaise may be present.

Imaging and labs are not necessary, but leukocytosis may suggest the presence of an abscess requiring drainage.

## Management

Asymptomatic cysts or sinuses do not require treatment.

Pilonidal abscess is treated with incision and drainage with removal of all hair and debris from the abscess cavity.

Antibiotics generally are not needed in the absence of surrounding cellulitis.

Patients often have recurrent infections and may develop draining fistulas.

For chronic or refractory disease, surgical excision is the definitive treatment.

## 26. PITYRIASIS ROSEA

Emily Singer, Tarlan Hedayati



**FIGURE 1.** Rash on the torso in a "Christmas" tree pattern.



**FIGURE 2.** Herald patch of pityriasis rosea.

## Clinical Presentation

A common, usually asymptomatic, rash most prevalent on the torso and proximal extremities

Often starts as a single round or oval “herald” patch that can mimic ringworm in appearance followed by more diffuse lesions 1 to 2 weeks later

A fine scale may be present along the circumference of the lesions.

## Diagnosis

Diagnosis is based on history and physical exam.

Can distinguish from secondary syphilis if herald patch is absent via syphilis serologic testing

Can mimic drug eruptions, nummular eczema, tinea, or viral exanthems

## Management

Benign and self-limited illness, thus no treatment is necessary

Direct sunlight may hasten the resolution of symptoms.

Extensive disease can be treated with prednisone 20 mg twice daily for 1 week.

## 27. PSORIASIS

Matthew Weeks, Jeffrey J. Schaider



FIGURE 1. Psoriasis.

### Clinical Presentation

Chronic, immune-mediated skin disorder, usually with erythematous papules and plaques with silver scaling, sometimes associated with pruritis. Plaques frequently occur on the extensor surfaces (elbows and knees) but can affect any area of the body.

Usual age of first presentation is in the fourth decade.

Associated with smoking and obesity

Commonly have family history of psoriasis or psoriatic arthritis

Symptoms tend to be more severe in patients with HIV.

### Diagnosis

Plaque psoriasis is the most common type, with round red patches with central plaques of silvery white scale.

Pustular psoriasis is a less common potentially life-threatening form. There is diffuse scaling, pustules, and eroding skin lesions.

Differential includes seborrheic dermatitis, lichen simplex chronicus, and atopic dermatitis. Occasionally, skin biopsy is needed to differentiate.

## Management

For mild disease, provide topical corticosteroids and emollients.

For severe disease, systemic methotrexate, retinoids, and immunomodulators are helpful. These are given only after consultation with a dermatologist.

If overlying suprainfection or cellulitis is present, antibiotics are indicated.

Referral to dermatologist for further outpatient treatment



## 28. PURPLE GLOVE SYNDROME

Mark Livak, John Bailitz



**FIGURE 1.** Purple glove syndrome. (From O'Brien TJ, Cascino GD, So EL, et al. Incidence and clinical consequence of the purple glove syndrome in patients receiving intravenous phenytoin. *Neurology*. 1998;51(4):1034-1039.)

### Clinical Presentation

Discoloration and edema evident at infusion site and distal limb after intravenous (IV) phenytoin infusion

Can occur with phenytoin extravasation; can also occur with functioning IV lines

Incidence is up to 6% of those receiving IV phenytoin.

Symptoms are more common in elderly, those receiving large or multiple doses of phenytoin, and those receiving phenytoin at higher infusion rates.

May progress to vascular compression, compartment syndrome, ischemia, and necrosis

### Diagnosis

Blue or purple discoloration begins around the IV insertion site 2 to 12 hours post-infusion.

Increasing edema and discoloration extends distal and proximal to IV site 12 to 24 hours post-infusion.

Discoloration and edema slowly resolve over the following days to weeks.

### Management

Mainstay of treatment is supportive care.

Provide analgesia.

Elevate and massage the affected limb.

Apply compression and gentle heat.

## 29. PYOGENIC GRANULOMA

Audrey Herbert, Deborah Kimball



FIGURE 1. Pyogenic granuloma of the finger.

### Clinical Presentation

Rapidly growing skin lesions that commonly occur on head, neck, hands, feet, and upper back

Generally appear as red nodules that easily bleed and rapidly progress over a period of weeks

Usually occur secondary to minor injury or trauma

Can arise during pregnancy due to hormonal changes

### Diagnosis

Diagnosis is based on history and physical exam.

Lesions contain small blood vessels, not pus as the name *pyogenic* would suggest.

Appearance is similar to amelanotic nodular melanoma; biopsy can be done if diagnosis is questionable.

## Management

Smaller lesions may resolve spontaneously and not need intervention.

Larger lesions are sometimes chemically treated or can be scraped off and cauterized to control bleeding.

Reoccurrences are common after removal.

Full-thickness excision performed by a dermatologist is the most definitive treatment.

## 30. RED MAN SYNDROME

Isam Nasr



**FIGURE 1.** Red man syndrome. Erythematous urticarial rash. (Reproduced from Nallasivan M, Maher F, Murthy K. Rare case of "red man" syndrome in a female patient treated with oral vancomycin for *Clostridium difficile* diarrhea. *BMJ Case Rep.* 2009;2009. doi:10.1136/bcr.03.2009.1705, with permission from BMJ Publishing Group Ltd.)

### Clinical Presentation

A rate-dependent infusion reaction, not a true allergic reaction

Most commonly seen with rapid infusion of the first dose of vancomycin but has been reported with ciprofloxacin, amphotericin B, rifampin, and teicoplanin

Red man syndrome (RMS) is characterized by flushing, erythema, and pruritus, usually affecting the upper body, neck, and face more than the lower body.

Pain and muscle spasms in the back and chest, dyspnea, and hypotension may also occur.

Combination of vancomycin with opioids, radiocontrast dye, and some muscle relaxants enhances dose- or rate-related mast cell degranulation leading to RMS.

## Diagnosis

Diagnosis is based on clinical presentation of symptoms, which may appear about 4 to 10 minutes after starting infusion or soon after its completion.

## Management

Discontinue infusion once symptoms occur, and administer a dose of diphenhydramine.

Infusion can be resumed at a slower rate and/or at a lesser dosage once the rash and itching dissipate.

Hypotension will require intravenous fluids, and, if severe, vasopressors may be needed.

Empiric premedication with antihistamines to prevent RMS is not usually necessary.



## 31. ROCKY MOUNTAIN SPOTTED FEVER

Neeraj Chhabra, Robert Feldman



**FIGURE 1.** Classic petechial rash of Rocky Mountain spotted fever. (From Burkhart C, Morrell D, Goldsmith LA, et al. *VisualDx: Essential Pediatric Dermatology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.)

### Clinical Presentation

A tick-borne systemic vasculitis caused by the bacteria *Rickettsia rickettsii*. Endemic to the southeastern United States, although cases have been reported throughout the contiguous United States.

Illness presents with the sudden onset of fever, headache, and myalgias with the rash appearing 2 to 4 days later.

Rash appearance is variable and may be atypical or absent in 20% of cases.

The initial erythematous maculopapular rash on the wrists and ankles evolves to the classic petechiae or ecchymoses with palm and sole involvement in only about half of all cases.

Complications include myocarditis, acute respiratory distress syndrome, renal failure, and disseminated intravascular coagulation.

### Diagnosis

Must be made on clinical grounds prior to confirmatory testing, although



clinical signs are highly variable

Nonspecific laboratory findings such as thrombocytopenia, anemia, and hyponatremia are reported but unreliable markers of disease.

Serology testing is used to confirm diagnosis after treatment but skin biopsy with immunofluorescent assay and staining may yield positive results as early as day 3 of illness.

Difficult to distinguish from meningococcemia and may require either workup or treatment for both diseases

## Management

Doxycycline is the first-line treatment for patients of all ages with chloramphenicol reserved for patients with pregnancy or life-threatening allergies.

Admission is indicated for all patients with a presumptive diagnosis unless otherwise advised by a specialist.

Early infectious disease consultation is recommended.

Mortality is linked to a delayed administration of antibiotics.

## 32. SCABIES

Michael J. Uguccioni, Jordan Moskoff



FIGURE 1. Scabies, wrist.



FIGURE 2. Scabies, magnified view.

### Clinical Presentation

Discrete, erythematous, papular rash caused by a mite

Typically acquired by sleeping in the bedding of infested individuals and associated with poor living conditions

Burrows may be visible (short, S-shaped tracks, 2 to 15 mm long, pink/white in color, and slightly elevated).

Lesions occur in crevasses (i.e., in between the fingers or toes, the buttocks,

elbow, genital area, and under the breasts in women) and typically spare the face, neck, palms, and soles.

Intensely pruritic, particularly at night; scratching may cause destruction of burrows.

## Diagnosis

Most commonly clinical, based on history and skin examination

If unclear, the physician may perform a skin scraping of a burrow or papule with subsequent microscopy looking for mites, eggs, or feces.

If burrows are difficult to visualize, the physician may use a felt-tip marker to draw across the rash and wipe afterward with alcohol. The ink penetrates deeper into the skin for enhancement of any burrows.

Vesicles are isolated, pinpoint, filled with serous rather than purulent fluid, and remain discrete (as opposed to poison ivy in which vesicles classically present in linear streaks).

## Management

The initial treatment of choice is permethrin 5% topical cream, which is applied from the neck to the soles of the feet and washed off 8 to 14 hours after application.

An alternative treatment for patients who cannot tolerate or are unlikely to comply with a topical regimen is oral ivermectin, given as a single dose of 200 µg/kg followed by a second dose 10 days later.

Antihistamines (i.e., hydroxyzine) are often used for symptomatic relief because scratching may result in secondary lesions or infection.

All sexual, close personal, and household contacts must be treated even if asymptomatic, and all recently worn clothing, towels, and bedding must be washed at 140°F or higher to lower the risk of reinfestation.

## 33A. SKIN CANCER: BASAL CELL CARCINOMA

Paul Bobryshev, Trevor Lewis



**FIGURE 1.** Cystic BCC. (Courtesy of Sumul Ashok Gandhi, MD.)



**FIGURE 2.** Nodular BCC. (Courtesy of Sumul Ashok Gandhi, MD.)



**FIGURE 3.** Pigmented BCC. (Courtesy of Sumul Ashok Gandhi, MD.)



**FIGURE 4.** Superficial BCC. (Courtesy of Sumul Ashok Gandhi, MD.)

## Clinical Presentation

Most common form of skin cancer

Cells arise from epidermis.

Most important risk factor is sun exposure; lesions usually appear in sun-exposed areas (head and neck).

Locally invasive without risk of distant metastasis

More common in fair-skinned patients

## Diagnosis

May be single or multiple

Usually painless

Distinct types of basal cell carcinoma include nodular, pigmented, cystic, superficial, micronodular, and morpheaform.

## Management

Suspicious lesions require biopsy, a procedure rarely done in emergency department.

Treat complications of visceral involvement by locally invasive BCC.

Patients are generally discharged with instructions on obtaining biopsy and/or further evaluation.



## 33B. SKIN CANCER: MELANOMA

Paul Bobryshev, Trevor Lewis



**FIGURE 1.** Malignant melanoma.



**FIGURE 2.** Nodular melanoma.

### Clinical Presentation

Five percent of all diagnosed skin cancer in United States

Seventy-five percent of skin cancer deaths  
Most important risk factor is sun exposure, especially sunburn.  
Risk of regional lymph node and distant metastasis

## Diagnosis

Pigmented skin lesion (2% will be amelanotic)  
Features suggestive of melanoma (the ABCDs of melanoma):  
Asymmetry (not regularly round or oval)  
Border irregularity (notched or poorly defined)  
Bleeding (spontaneous)  
Color variation (shades or combination of brown, tan, red, white, or blue-black)  
Diameter >6 mm

## Management

Suspicious lesions require biopsy, a procedure rarely done in emergency department.  
Patients are generally discharged with instructions on obtaining biopsy and/or further evaluation.  
Chest x-ray may show pulmonary involvement by metastatic melanoma.  
Head or body computed tomography scan may show visceral involvement by metastatic melanoma.



## 33C. SKIN CANCER: SQUAMOUS CELL CARCINOMA

Paul Bobryshev, Trevor Lewis



**FIGURE 1.** Invasive squamous cell carcinoma of the hand.



**FIGURE 2.** Invasive squamous cell carcinoma of the face.

### Clinical Presentation

Second most common skin cancer

Most important risk factor is sun exposure, especially sunburn.

Seventy percent occur on head and neck.

Risk of regional lymph node and distant metastasis  
Squamous cell carcinoma (SCC) lesions of mucosal surfaces are more aggressive.  
SCC often associated with human papilloma virus

## Diagnosis

Characteristic lesion is raised, firm, keratotic papule or plaque.  
Often enlarging  
Usually asymptomatic but may be ulcerated and painful  
Ulcers often crust and ooze.  
Cranial nerve involvement may indicate an aggressive tumor with perineural invasion (facial numbness, asymmetry, weakness, or pain).

## Management

Suspicious lesions require biopsy, a procedure rarely done in emergency department.  
Patients are generally discharged with instructions on obtaining biopsy and/or further evaluation.

## 34. STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Matthew Weeks, Jeffrey J. Schaider



**FIGURE 1.** Staphylococcal scalded skin syndrome. (From Burkhardt C, Morell D, Goldsmith LA, et al. *VisualDx: Essential Pediatric Dermatology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2009, with permission.)



**FIGURE 2.** Staphylococcal scalded skin syndrome. Note the scalded appearance of the skin under the ruptured bullae of the chest and axilla in this child with staphylococcal scalded skin syndrome. (From Lippincott's Nursing Advisor. Philadelphia, PA: Nursing Solutions; 2014.)

### Clinical Presentation

Caused by exfoliative, epidermolytic toxins of some strains of *Staphylococcus aureus*

Most common in children and neonates but much higher mortality in adults

Most complications result from sepsis and suprainfection.

## Diagnosis

Often starts as a localized *S. aureus* infection, progressing to macular erythematous rash either locally or at distant site, with epidermal exfoliation. Initially, patient may not appear toxic.

Bullae often develop, and Nikolsky's sign is present.

Differential includes Stevens-Johnson syndrome, toxic epidermal necrolysis (TEN), toxic shock syndrome, and scarlet fever.

Mucous membranes are usually spared in staphylococcal scalded skin syndrome (SSSS), whereas in TEN there is usually involvement of these sites (i.e., mouth, anus, and vagina).

In SSSS, toxins travel hematogenously to distant sites, whereas in bullous impetigo, the toxins remain at the area of infection.

## Management

Maintain hydration with intravenous (IV) fluids.

Wound care similar to that provided to burn patients.

Initiate IV antibiotics to cover for presumed methicillin-resistant *Staphylococcus aureus*.

Avoid steroids. Also avoid nonsteroidal antiinflammatory drugs due to renal impairment.

Consultants include burn service, dermatology, infectious disease.

Toxic patients will require intensive care unit admission.

## 35. STEVENS-JOHNSON SYNDROME

Jason Lee, Mark P. Kling



**FIGURE 1.** Mucous membrane involvement in a patient with Stevens-Johnson syndrome. (From Domino FJ, ed. *The 5-Minute Clinical Consult* 2015. Philadelphia, PA: Lippincott Williams & Wilkins; 2014.)

### Clinical Presentation

A hypersensitivity reaction historically thought to be a severe form of erythema multiforme (EM)

More than one mucosal surface is involved in Stevens-Johnson syndrome (SJS), differentiating it from EM.

Drugs including antibiotics, nonsteroidal antiinflammatory drugs, and anticonvulsants are more common precipitants than infectious causes, such as mycoplasma or HIV.

May present with typical targetoid lesions, when <10% body surface area is involved

Unlike the raised targetoid/iris lesions of EM, SJS lesions are more likely to be flat and macular with or without blisters or bullae; ocular involvement is common.

## Diagnosis

Based on history and physical exam

The extent of body surface area involved distinguishes SJS from toxic epidermal necrolysis (TEN). Less than 10% total body surface involvement is categorized as SJS. Diagnosis overlaps with TEN when 10% to 30% of total body surface area is involved.

## Management

Identify the precipitant and discontinue the causative medication.

Supportive care is the mainstay of initial management.

SJS is a medical emergency warranting admission and consultation with dermatology and a burn surgeon.

Ophthalmology consultation for ocular involvement, which is common in SJS



## 36. THROMBOCYTOPENIA

Marcus Emebo, Jenny Lu



**FIGURE 1.** Petechiae on the legs.



**FIGURE 2.** Hemorrhagic bullae of the mucus membranes in a patient with a platelet count of  $7 \times 10^9/L$ .

### Clinical Presentation

Defined as a platelet count  $<150 \times 10^9/L$

Results in defect in primary hemostasis

May manifest with cutaneous or mucosal petechiae, nonpalpable purpura, epistaxis, menorrhagia, gastrointestinal bleeding, or bleeding complications



after surgery or dental extractions

Clinically significant spontaneous bleeding usually only occurs when platelet count is  $<5$  to  $10 \times 10^9/L$ .

Mechanisms include decreased bone marrow production (e.g., aplastic anemia), splenic sequestration (e.g., splenomegaly), and increased destruction (e.g., immune thrombocytopenia [ITP], thrombotic thrombocytopenic purpura [TTP]).

## Diagnosis

Obtain complete medical history, specifically prior blood transfusions, recent medications (nonsteroidal antiinflammatory drugs, heparin), family history of bleeding disorders, and social history (alcohol abuse).

Complete blood count with differential is helpful to evaluate blood cell lines.

Obtain a peripheral blood smear.

Consider evaluating liver and renal function, coagulation profile including D-dimer, and studies for hemolysis.

## Management

Avoid giving medications with antiplatelet activity.

Avoid prophylactic platelet transfusion in suspected or confirmed heparin-induced thrombocytopenia, TTP, or HUS, as these patients are at risk for platelet-mediated thrombotic complications.

Any life- or limb-threatening bleeding should receive platelet transfusion regardless of the underlying etiology.

Emergent bleeding in ITP warrants combination of corticosteroids with intravenous immunoglobulin to promote rapid increase in platelets.

Consult hematology for possible emergent plasma exchange in TTP.

Prophylactic platelet transfusion is recommended for patients with anticipated risk of bleeding from trauma, minor procedures, or surgery. The platelet goal under these circumstances is  $>50 \times 10^9/L$ .

## 37. TINEA CAPITIS

Emily Singer, Tarlan Hedayati



**FIGURE 1.** Tinea capitis. (From Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)



**FIGURE 2.** Tinea capitis with alopecia. (From Goodheart HP. *Goodheart's Photoguide of Common Skin Disorders*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.)

### Clinical Presentation

Tinea burrows into the hair follicle and grows within the hair shaft. It can cause an inflammatory or noninflammatory response.

Usually occurs in children, most frequently ages 3 to 7 years

Unlike other forms of tinea, tinea capitis is contagious via close personal contact and contaminated clothing.

Full range of presentations includes diffuse scaling of the scalp similar to dandruff, patchy/scaly alopecia, and diffuse scattered pustules.

## Diagnosis

Diagnosis is based on history and physical exam.

Both inflammatory and noninflammatory varieties start as a patch of hair loss with a fine scale.

Noninflammatory variety presents with either hair broken off the scalp or with short stubs of broken hairs.

Kerion is a form of tinea capitis with severe inflammatory response; presents with boggy induration of the scalp

Diagnosis can be confirmed via potassium hydroxide preparation of plucked hairs.

## Management

Topical treatment alone is ineffective but can be used with oral therapy to shorten duration of treatment and reduce the risk of spreading to others.

Griseofulvin is the drug of choice in children, as it is safe and well-tolerated.

Length of treatment varies based on organism but usually is 8 to 10 weeks.

Alternative treatments include terbinafine, itraconazole, fluconazole, and ketoconazole.

## 38. TINEA CORPORIS

Emily Singer, Tarlan Hedayati



**FIGURE 1.** Tinea corporis. (From Burkhart C, Morrell D, Goldsmith LA, et al. *VisualDx: Essential Pediatric Dermatology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.)

### Clinical Presentation

Tinea infection of the face or body excluding the groin

Also known as *ringworm* due to its characteristic ringlike lesions

Lesions start as flat, scaly macules that develop a raised edge and grow outward with the scale, most prominent along the border with central clearing. Severity and level of inflammation depends on type of fungus, patient immune response, and presence or absence of bacterial overgrowth.

### Diagnosis

Diagnosis is based on history and physical exam.

Distinguish from pityriasis rosea by the close proximity of the scale to the

edge of the lesion

If needed, the type of fungus can be determined by a scraping of the scale.

## Management

Treat with topical antifungal agents.

Generally responds after 2 weeks of treatment but continue therapy for 1 week after resolution of symptoms

Extensive infection can be treated orally with fluconazole.

Deep inflammatory lesions may require multiple months of oral therapy for complete resolution.

Short course of oral steroids may be considered for patients with severely inflamed lesions.



## 39. TINEA CRURIS

Emily Singer, Tarlan Hedayati



FIGURE 1. Tinea cruris.

### Clinical Presentation

Informally known as *jock itch*

Like all tinea, risk factor is a warm, moist environment in which to grow.

Tinea cruris flourishes in patients with large skinfolds that allow moisture to accumulate.

More common in men than women but can affect either gender

### Diagnosis

Symptoms begin in the crural fold and can be unilateral or bilateral, then advance from the crural fold onto the thigh with an erythematous base and well-defined scale.

Scrotal involvement is rare, which distinguishes tinea cruris from a candidal infection.

Diagnosis is based on history and physical exam but can be confirmed with

potassium hydroxide preparation of specimen taken from scaling border.

## Management

Infection is treated with topical antifungal cream, given twice daily. Therapy should be continued for at least 10 days, even if rapid improvement occurs.

Treatment with topical steroid creams exacerbates symptoms.

Severe or persistent cases may require oral therapy with fluconazole.

Nonmedicated drying powders help prevent recurrence once acute episode has resolved.



## 40. TINEA PEDIS

Emily Singer, Tarlan Hedayati



FIGURE 1. Tinea pedis.

### Clinical Presentation

The foot is the area most commonly affected by tinea infection due to the warm, moist environment inside shoes.

For interdigital infections, the web space between toes becomes either white, wet, and macerated or scaly, dry, and fissured. Affected area may be pruritic. The infection can then spread to the top or sole of the foot with a typical scaly border.

A highly inflammatory response can develop in patients with tinea, causing vesicles that may rupture and become superinfected. A second wave of vesicles can occur at distant sites due to an allergic-type reaction to the fungus. The allergic response resolves with control of the infection.

### Diagnosis

Diagnosis is based on history and physical exam.

Fungal culture can be sent for definitive diagnosis in atypical presentations.

## Management

Over-the-counter terbinafine 1% cream twice daily for 1 week achieves good cure rates for interdigital tinea pedis.

Fungistatic agents (e.g., clotrimazole) take much longer to work (require 4 weeks of treatment) and have a higher rate of treatment failure.

Recurrence can be avoided by keeping the feet, including the interdigital space, dry.

## 41. TINEA VERSICOLOR

Emily Singer, Tarlan Hedayati



**FIGURE 1.** Tinea versicolor of the back.



**FIGURE 2.** Tinea versicolor of the arm.

### Clinical Presentation

Common superficial fungal infection of the skin

Starts as multiple small, circular macules that enlarge and coalesce. Macules

can present in a variety of colors ranging from hypo- to hyperpigmented. An affected individual has lesions of a single color.

Hypopigmented lesions in light-skinned individuals become more apparent when the skin tans.

Color change is due to melanocyte damage by the fungal species.

Rash is sometimes pruritic but usually asymptomatic.

## Diagnosis

Diagnosis is mainly clinical, based on characteristic appearance.

Diagnosis is based on history and physical exam but can be confirmed with potassium hydroxide preparation of scraping.

Scrapings fluoresce pale yellow to white under Wood lamp.

Fungal culture is possible but not usually needed.

## Management

Topical treatment can be tried if only a small area is affected. Ketoconazole shampoo either once or daily for 3 days is first line. One can also try selenium sulfide suspension, terbinafine solution, or azole creams.

Oral treatment should be used for patients with extensive disease or who failed topical therapy. Ketoconazole, itraconazole, or fluconazole are all effective.

Oral terbinafine or griseofulvin is not effective.

Absence of fine scale using a no. 15 blade indicates the fungus has been eliminated, but altered pigmentation persists after the fungus has been eradicated. Sunlight accelerates improvement in pigmentation.

Recurrence is common and occurs in 40% to 60% of patients.

## 42. TOXIC EPIDERMAL NECROLYSIS

Jason Lee, Mark P. Kling



**FIGURE 1.** Mucous membrane involvement of toxic epidermal necrolysis. (Image courtesy of Ashit Marwah, MD. From Goodheart HP. *Goodheart's Photoguide to Common Skin Disorders: Diagnosis and Management*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.)



**FIGURE 2.** Skin lesion in toxic epidermal necrolysis. (From Mulholland MW, Lillemoe KD, Doherty GM, et al. *Greenfield's Surgery: Scientific Principles And Practice*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.)

### Clinical Presentation

A hypersensitivity reaction considered a more severe variant of Stevens-Johnson syndrome (SJS) with similar precipitants

Medications including anticonvulsants, antibiotics, and nonsteroidal antiinflammatory drugs are the most common cause.

Skin lesions may range from tender erythematous lesions in an early presentation to intact bullae or open exfoliated lesions later in the course of the disease. Bullae and blisters demonstrate a positive Nikolsky's sign.



Two or more mucous membranes are involved; ocular involvement is common.

## Diagnosis

Based on history and physical exam

Degree of total body surface area (TBSA) involvement differentiates toxic epidermal necrolysis (TEN) from SJS. More than 30% TBSA involvement is categorized as TEN. Diagnosis overlaps with SJS when 10% to 30% of TBSA is involved.

Laboratory testing is indicated for the evaluation of electrolyte imbalances and possible sources of infection.

## Management

Discontinue any precipitating medications.

Manage airway, breathing, and circulations; treatment is similar to that of a burn patient with fluid resuscitation based on institutional burn management protocols.

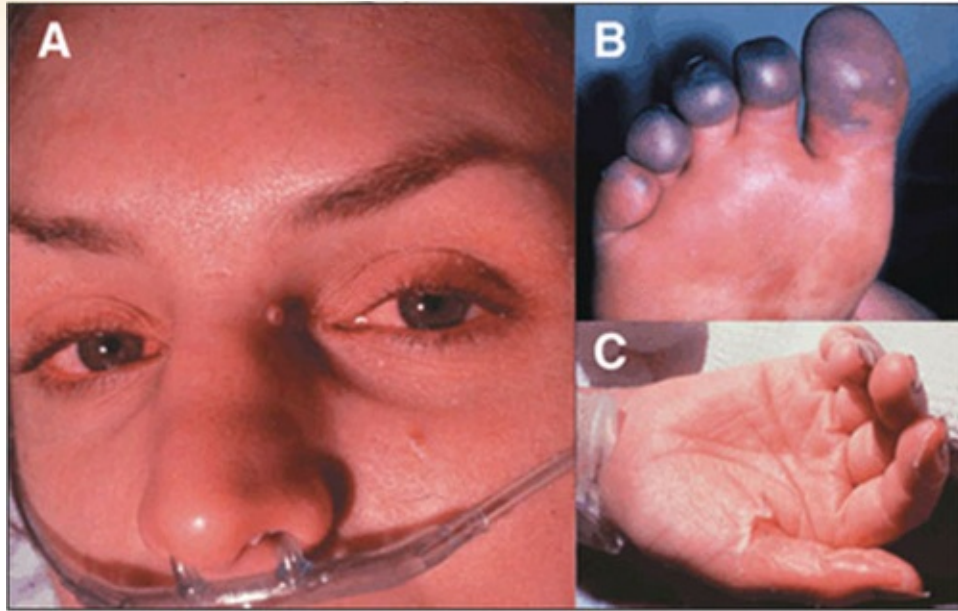
Provide appropriate burn/wound care; manage impaired thermoregulation, electrolyte imbalances; identify and treat precipitating or resultant infections.

Consult dermatology and a burn surgeon for further management. Consult ophthalmologist for management of ocular complications.

Admit to a critical care bed or ideally a burn unit for further care.

## 43. TOXIC SHOCK SYNDROME

Neeraj Chhabra, Robert Feldman



**FIGURE 1.** Toxic shock syndrome. **A:** Appearance of the rash associated with staphylococcal toxic shock syndrome (TSS). **B:** Gangrenous toes associated with prolonged hypotension in TSS. **C:** Desquamation of the skin that occurs during the resolution of TSS. (From Engleberg NC, Dermody T, DiRita V. *Schaechter's Mechanisms of Microbial Disease*. 5th ed. Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

Presents with the rapid onset of fever followed by rash, multisystem failure, and shock

Skin findings include a painless erythroderma resembling a sunburn that will desquamate in 10 to 14 days.

Rash can involve the palms, soles, and mucous membranes.

Traditional sources of infection include superabsorbent tampons, nasal packing, postoperative wounds, or pharynx.

### Diagnosis

Requires the presence of fever, shock, and multisystem involvement

Worsening of renal function and hemoglobinuria are present in almost all cases and may precede shock.

A thorough search for a source of infection must be done focusing on foreign



bodies, mucous membranes, and respiratory system.

Can present similarly to staphylococcal scalded skin syndrome, which spares mucous membranes, and Stevens-Johnson syndrome/toxic epidermal necrolysis, which may have an inciting exposure

## Management

Removal of bacterial sources (tampons, nasal packing, or other foreign bodies) and initiation of broad-spectrum antibiotics, including vancomycin, are the mainstays of treatment.

Adjunctive clindamycin may suppress synthesis of the bacterial toxin and facilitate phagocytosis of group A streptococci.

Surgical consultation may be necessary to remove infectious focus.

All patients should be admitted to a critical care setting with infectious disease consultation.

## 44. URTICARIA

Lisa R. Palivos



**FIGURE 1.** Urticaria of the back.



**FIGURE 2.** Urticaria of the arm.

## Clinical Presentation

Urticaria are pruritic, transient, edematous plaques with pale centers and raised borders.

Most commonly caused by medications

Can also be caused by infections, environmental or physical factors, pregnancy, malignancies, or foods

Urticaria can progress to angioedema or anaphylaxis.

## Diagnosis

Diagnosis is based on history and physical exam.

## Management

Treatment depends on severity and extent of reaction.

Epinephrine intramuscular or intravenous is the first-line treatment for anaphylaxis.

Second-line treatment consists of antihistamines (both  $H_1$  and  $H_2$  blockers)

and corticosteroids.

Bronchospasm can be treated with albuterol.

Identify and eliminate the inciting factor.

## 45. WARFARIN-INDUCED SKIN NECROSIS

Rosaura Fernandez



**FIGURE 1.** Warfarin-induced skin necrosis of the legs. (With kind permission from Springer Science+Business Media: Kozac N, Schattner A. Warfarin-induced skin necrosis. *J Gen Intern Med.* 2014;29[1]:248-249, Fig. 1.)

### Clinical Presentation

Rare condition, with prevalence of up to 0.1% of patients being treated with warfarin

Typical onset is 7 to 10 days after starting the medication.

High-risk groups are obese, premenopausal women actively being treated with warfarin for thromboembolic disease. Other populations who may be at an increased risk include those with underlying thrombophilia, such as protein C and protein S deficiencies.

Patients commonly present with spontaneous development of tender erythematous skin changes followed by underlying edema with rapid progression to hemorrhagic bullae and necrosis of the underlying subcutaneous tissue.

Areas more commonly affected are those with more subcutaneous fat such as the breast, abdomen, extremities, and buttocks and the penis in males.

If condition is advanced, the patient may have extensive necrosis.

### Diagnosis

Initial diagnosis is based on history and physical exam.

Diagnosis is supported by skin biopsy.

## Management

Warfarin should be immediately discontinued.

Patient should receive vitamin K and fresh frozen plasma to reverse the effects of warfarin, along with an infusion of heparin at therapeutic doses.

Management typically involves surgical debridement and, if necessary, amputation of involved areas.

If the involved area is significant, the patient may present with clinical signs of sepsis or septic shock.

Supportive care as clinically indicated.



SECTION

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ORTHOPEDIC

SECTION EDITOR

Scott C. Sherman



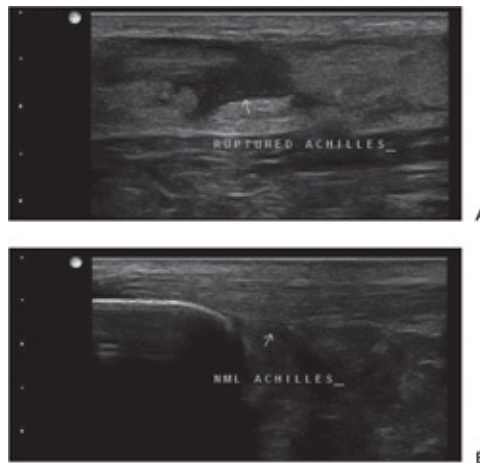
# 1. ACHILLES TENDON RUPTURE



Douglas Brtalik, Manoj Pariyadath



**FIGURE 1.** Achilles tendon rupture on the right. Note the loss of plantarflexion.



**FIGURE 2.** Ultrasound. **A:** Achilles tendon rupture. **B:** Normal Achilles tendon.

## Clinical Presentation

Acute rupture of the Achilles tendon is usually secondary to either forced dorsiflexion of an already plantar-flexed foot or sudden plantar flexion of a weight-bearing foot while the knee is extended.

It is the third most commonly ruptured tendon in the human body.

Most common in men between ages 30 to 40 years, with risk factors being

failure to warm-up or stretch prior to high-demand exercise, fluoroquinolone use, prior steroid injections in the area, and prior Achilles tendon rupture. The patient will often present with immediate inability to bear weight of the affected extremity, with large amount of swelling, and often a palpable defect in the posterior heel, where the Achilles tendon lies.

## Diagnosis

Plain radiographs are not helpful and are frequently normal in acute Achilles tendon rupture.

Diagnosis is frequently made with exam, with the patient holding the affected ankle in slight dorsiflexion, along with a weakness in plantar flexion, and a positive Thompson's test.

Thompson's test is performed by manually squeezing the patient's calf, while he or she is kneeling or prone, and observing for passive plantar flexion of the ankle. Lack of plantar flexion or significant asymmetry from the unaffected side equate to a positive Thompson's test.

Magnetic resonance imaging is the gold standard for diagnosis of acute Achilles tendon rupture, but diagnosis can also be made by other imaging modalities, most notably ultrasonography.

## Management

A posterior ankle splint should be placed with the ankle in a moderate degree of plantar flexion (gravity equinus position).

Achilles tendon rupture can be managed both operatively and nonoperatively. Operative management is associated with lower risk of tendon rerupture (<3%) but has increased risks of deep venous thrombosis, wound infection, and sural nerve damage.

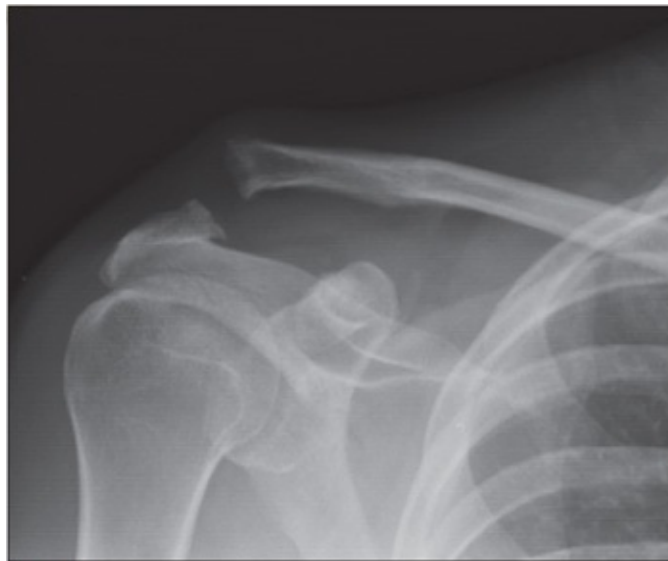
Nonoperative management has higher risks of tendon rerupture (10% to 12%), but newer studies have shown that early range of motion rehabilitation exercises can significantly decrease the risk of rerupture while avoiding increased risks of operative management listed earlier.

## **2. ACROMIOCLAVICULAR JOINT DISLOCATION**

Michael Gottlieb



**FIGURE 1.** Clinical photo of AC separation.



**FIGURE 2.** Radiograph of a third degree AC separation.

**Clinical Presentation**

The clavicle is secured to the scapula via the acromioclavicular (AC), coracoacromial (CA), and coracoclavicular (CC) ligaments.

AC joint injuries are typically caused by a direct fall onto the shoulder with an adducted arm.

Less commonly, an indirect mechanism occurs through a fall onto an outstretched arm, wherein the humeral head displaces the acromion superiorly.

The injury is classified by the Rockwood criteria:

Grade 1 is an AC joint sprain with no radiographic abnormalities.

Grade 2 is a complete tear of the AC ligament with widening of the AC gap on x-ray (normal AC gap  $\leq 4$  mm).

Grade 3 is a tear of the AC, CA, and CC ligaments with 100% displacement on x-ray.

Grade 4, 5, and 6 injuries are less common but occur when the clavicle dislocates posteriorly through the trapezius, superiorly above the acromion, or inferiorly below the coracoid process, respectively.

## Diagnosis

The diagnosis is made based on a combination of the clinical examination and standard shoulder radiographs.

The patient will typically have acute, localized pain at the AC joint with restricted range of motion.

In higher grade injuries, the clavicle may appear prominently, with loss of the normal shoulder contour.

## Management

Grade 1 to 3 injuries do not require surgery and can be managed with a sling for the first 1 to 2 weeks, early physical therapy, and pain control.

Grades 4 to 6 injuries require early surgical reduction and fixation.

### 3. ANKLE FRACTURE-DISLOCATION



Michael D. Zwank



**FIGURE 1.** Lateral ankle fracture-dislocation.



**FIGURE 2.** Anteroposterior (A) and lateral (B) ankle radiographs, demonstrating a posterolateral ankle fracture dislocation.

#### Clinical Presentation

Patient will be nonambulatory with obvious deformity of ankle joint.  
Can cause vascular compromise of foot as well as rapid skin necrosis from pressure/tenting of skin over bone  
Typically involves bi- or trimalleolar (medial, lateral, and posterior malleoli) fracture with associated dislocation

#### Diagnosis

Significant injury and deformity is apparent on physical exam.  
Rapid evaluation and diagnosis is important to help minimize risk of skin and

soft tissue injury.

Three-view ankle x-ray defines extent of injury.

May consider tibia/fibula x-rays if suspicion for associated proximal injury

## Management

Rapid reduction is critical to prevent skin necrosis from bone tenting.

Reduction should be performed prior to x-rays if delay is anticipated or prior to any interhospital transfer.

Reduction can be aided by local ankle block, ultrasound-guided popliteal sciatic nerve block, or procedural sedation.

After reduction, place posterior short leg and stirrup splints.

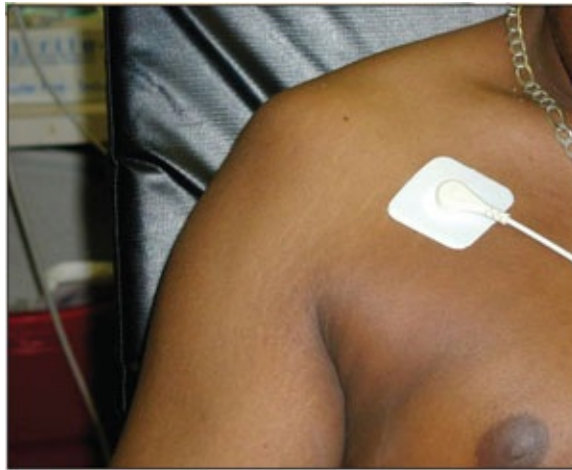
Can be safely discharged if adequate reduction

Early orthopedic follow-up is important, as nearly all will need definitive surgical management.



## 4. ANTERIOR SHOULDER DISLOCATION

Kraftin Schreyer, David A. Wald



**FIGURE 1.** Loss of rounded appearance of an anterior shoulder dislocation.



**FIGURE 2.** AP radiograph.

### Clinical Presentation

The shoulder is the most commonly dislocated major joint. Anterior dislocations account for 95% of all shoulder dislocations. The majority of these injuries occur during athletic activity.

A traumatic anterior shoulder dislocation typically occurs as a result of a



forceful blow to the abducted, externally rotated, and extended arm (blocking a basketball shot), a blow to the posterior upper arm, or a fall on an outstretched arm.

Chronic or recurrent dislocations can occur after trivial trauma or simply an exaggerated range of motion.

Patients typically present with a slightly abducted and externally rotated arm, a prominent acromion, and loss of the “rounded” appearance of a normal shoulder. It is common for patients to be supporting the injured extremity by holding it close to the body.

A careful evaluation to identify damage to surrounding neurovascular structures (i.e., axillary nerve injury) should be performed and documented pre- and postreduction.

## Diagnosis

The diagnosis is often suspected based on the mechanism of injury and the physical examination.

Plain radiographs (anteroposterior [AP], axillary lateral and the scapular “Y” view) should be obtained to confirm the dislocation and to exclude a fracture-dislocation. Up to 10% of acute traumatic anterior shoulder dislocations are associated with fractures, including greater tuberosity, Hill-Sachs, or Bankart fractures.

In patients with recurrent, atraumatic shoulder dislocation, radiographs may be avoided.

## Management

Anterior shoulder dislocations may be reduced by external rotation, scapular manipulation, prone positioning, and/or traction/countertraction.

Patients often require analgesia and sedation to facilitate the reduction.

Orthopedic consultation should be sought for patients who cannot be reduced in the emergency department or for those with an associated surgical neck humeral fracture.

Following a traumatic anterior shoulder dislocation, the shoulder is commonly immobilized for a brief period (2 weeks) prior to starting physical therapy.



## 5. AVASCULAR NECROSIS OF THE HIP

Ashli Burns, Andrew King



**FIGURE 1.** Radiograph of advanced avascular necrosis.

### Clinical Presentation

Pain with weight-bearing located primarily in the groin that can radiate to the buttock, knee, or thigh

Decreased range of motion of the hip joint due to pain; passive internal rotation of the hip is particularly painful.

Most commonly caused by previous trauma (femoral neck fractures and hip dislocations), which causes a disruption of blood flow to the femoral head; also seen in systemic lupus erythematosus, sickle cell disease, and chronic steroid use

### Diagnosis

Hip radiographs are the initial test of choice. Both “frog-leg” (with hip flexed

and externally rotated) and anteroposterior views should be obtained. Radiographs of the unaffected hip can be a helpful comparison. X-rays have poor sensitivity in early avascular necrosis; therefore, magnetic resonance imaging (MRI) should be obtained if there is high clinical suspicion. MRI is the most sensitive imaging modality and is the gold standard for diagnosis.

## Management

Initial management includes aggressive pain control and no weight bearing on the affected leg. Close orthopedic follow-up will be necessary, as surgical fixation is the definitive treatment.

## 6. BENNETT'S AND ROLANDO'S FRACTURE

Basem F. Khishfe



FIGURE 1. Bennett's fracture.



FIGURE 2. Rolando's fracture.

### Clinical Presentation

Two types of intra-articular 1st metacarpal base fractures exist. Bennett's fracture is a fracture with subluxation of the 1st metacarpal. Rolando's fracture is a comminuted fracture involving the joint. The most common mechanism is an axial force directed against the first metacarpal.

## Diagnosis

Pain, swelling, and tenderness is noted at the base of the 1st metacarpal. Thumb radiographs are usually sufficient to identify the fracture.

## Management

Bennett's fracture is usually managed by reduction and thumb spica splint. Surgery is indicated if there is involvement of >25% of the articular surface and the fracture is >1 to 2 mm displaced.

Rolando's fracture has a worse prognosis. Definitive treatment involves open reduction internal fixation or external fixation depending on the size of the bone fragments.

Traumatic arthritis is one of the most common complications of these fractures.

## 7. BIMALLEOLAR FRACTURES

John Cook



**FIGURE 1.** Bimalleolar fracture.





**FIGURE 2.** Functional bimalleolar fracture.

## Clinical Presentation

A fracture of the medial and lateral malleoli is termed a *bimalleolar fracture*. A fracture of the lateral malleolus with a ruptured deltoid ligament, with an intact medial malleolus, is known as a *functional bimalleolar fracture* or *bimalleolar equivalent fracture*.

Usually due to rotational forces, these injuries represent 25% of ankle fractures.

On examination, gross ankle soft tissue swelling is noted.

## Diagnosis

Anteroposterior (AP), lateral, and mortise views of the ankle

A “functional” bimalleolar fracture is diagnosed by measuring the medial clear space (distance between the talus and medial malleolus) on the AP radiograph. Normally, the medial clear space is  $<4$  mm.

An external rotation stress film can reveal a ruptured deltoid ligament in a functional bimalleolar fracture.

## Management

Perform careful examination of neurovascular status. Note any soft tissue injury or skin blistering.

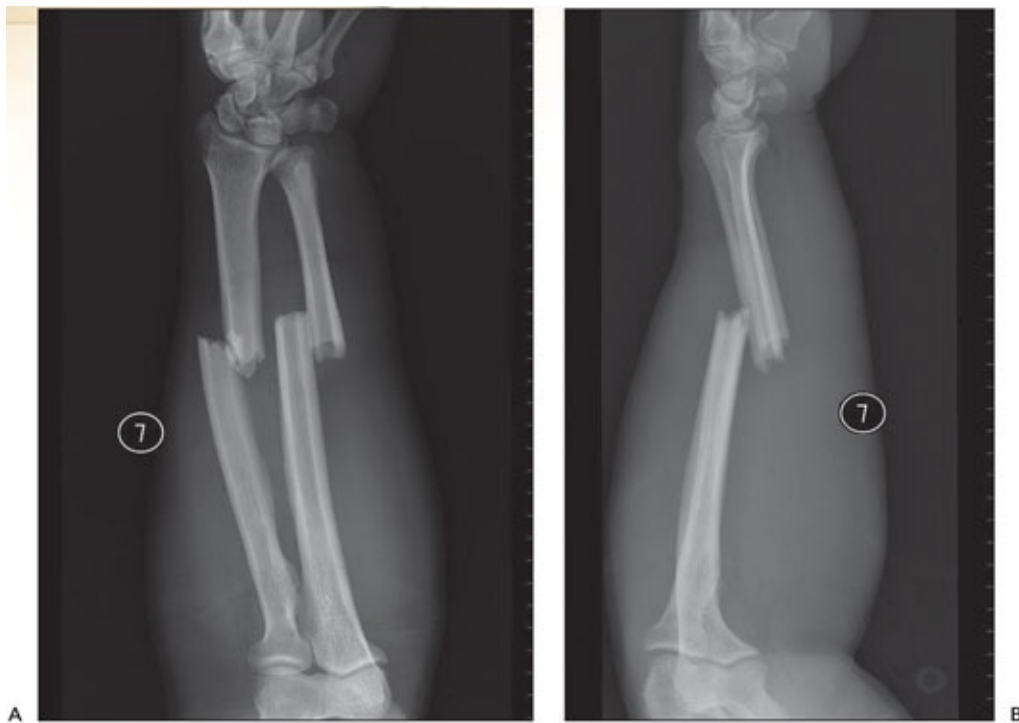
Fracture reduction with appropriate analgesia or procedural sedation in the emergency department; posterior splint combined with a U-shaped splint should be applied followed by postreduction films.

This injury is most often managed with surgery.

Nonoperative management is acceptable in the setting of nondisplaced, stable fractures with no disruption of the syndesmosis or following stable anatomic reduction.

## 8. BOTH BONE FOREARM FRACTURE

Michael J. Uguccioni



**FIGURE 1.** Forearm fracture. AP (A) and lateral (B) views.

### Clinical Presentation

Fractures of both the radius and ulnar shafts

Classification is based on bone displacement and angulation (and association with radial head dislocation).

The most common mechanism for this condition is a direct blow to the forearm and, in children, from a fall on an outstretched arm.

### Diagnosis

Physical examination will typically reveal pain, edema, and diminished hand and forearm function. An obvious forearm deformity is typically present. With open fractures, the physical exam may illicit deficits of the radial,

median, or ulnar nerves (nerve deficits are uncommon in closed injuries). Plain radiographs are sufficient to make this diagnosis. Wrist and elbow views should also be obtained to evaluate for associated injury to the proximal and distal radioulnar joints.

## Management

Nondisplaced both bone forearm fractures, although uncommon, are treated with anteroposterior (AP) splinting and subsequent long arm casting. Repeat imaging is necessary, as delayed displacement is fairly common.

Displaced fractures are rarely successfully managed with closed reduction in adults, and these patients are placed in long arm immobilization and referred for operative management. Closed reduction does achieve good results in most pediatric cases. Open fractures require immediate operative intervention by an orthopedic surgeon.

Fractures associated with radial head dislocation require open reduction and internal fixation.

Greenstick fractures are treated with long arm splinting and possibly referred to an orthopedic surgeon. Reduction of these injuries may be required if angulation exceeds 15°.

## 9. BOXER'S FRACTURE

Sierra Beck



**FIGURE 1.** Anteroposterior radiograph.



**FIGURE 2.** Oblique radiograph.

## Clinical Presentation

Fractures of the 4th or 5th metacarpal neck typically occur as a result of direct impact to a closed fist.

Physical exam shows depression of the affected knuckle and soft tissue swelling over the fracture site on the dorsum of the hand.

Look for puncture wound over the metacarpophalangeal (MCP) joint, which may indicate a “fight bite.”

## Diagnosis

Radiographs of the hand should include a true lateral to determine the degree of angulation of the distal fragment. The metacarpal head will typically displace toward the palm.

Physical exam should include passive flexion of the fingers to evaluate for “scissoring” of the fingers, which indicates rotational deformity and need for reduction.

Assess for associated digital nerve, extensor tendon, or ligamentous injury.

## Management

Reduction should be performed if there is  $>35^{\circ}$  of angulation in the ring finger or  $>45^{\circ}$  of angulation in the small finger. Any rotational deformity requires reduction.

Reduction is typically performed under hematoma or ulnar nerve block.

Traction is applied to the metacarpal. The MCP and interphalangeal joints are held in  $90^{\circ}$  of flexion while pressure is applied on the dorsal surface over the proximal metacarpal shaft and on the volar surface of the flexed proximal interphalangeal (PIP) joint.

Fractures should be immobilized with an ulnar gutter splint extending from below the elbow to just below the PIP joints. The metacarpals should be splinted at  $70^{\circ}$  to  $90^{\circ}$  of flexion. Early orthopedic referral is indicated, as fractures often fail closed reduction and may require operative management.



## 10. CALCANEUS FRACTURE

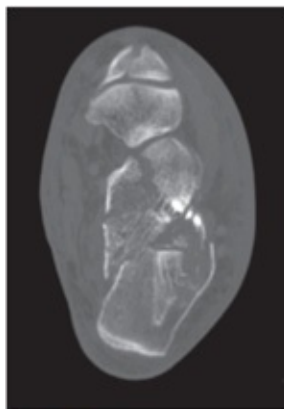
Stacey Chamberlain



**FIGURE 1.** Mondor's sign.



**FIGURE 2.** Plain radiograph. Note the inconsistent density within the body of the calcaneus and disruption of the trabecular pattern of the bone.



**FIGURE 3.** CT scan.

Clinical Presentation

Calcaneus fractures account for 60% of tarsal bone fractures. Fractures are usually closed, and 70% are intra-articular.

The most common mechanism for a calcaneus fracture is a fall from height landing on one's feet (axial loading).

Patients should be evaluated for concomitant injuries associated with a fall from height such as vertebral fractures (found in 10% to 15%), soft tissue injuries including the Achilles tendon, and contralateral fractures (7%).

## Diagnosis

Suggested by tenderness elicited with squeezing the patient's heel

Hematoma extending to the sole of the foot (Mondor's sign) is pathognomonic.

The Böhler's angle is used to detect subtle fractures on a lateral radiograph; an angle  $<20^\circ$  suggests a possible fracture.

Computed tomography (CT) is the study of choice for classifying fractures and determining the degree of comminution and displacement.

## Management

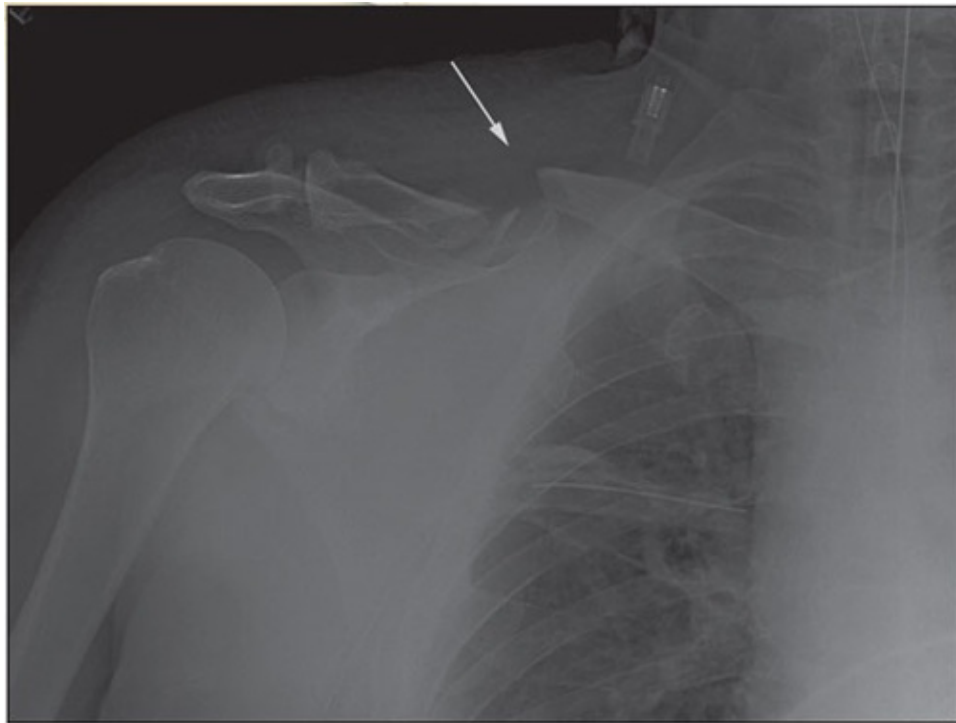
A postmold splint or bulky compression dressing should be applied, and patients should be non-weight-bearing for at least 2 weeks with close orthopedic follow-up.

Most extraarticular fractures are managed nonoperatively with a short leg cast, non-weight-bearing for 2 to 3 weeks, range-of-motion exercises, and progressive weight-bearing for 2 to 3 months.

Displaced intraarticular fractures require closed reduction with percutaneous fixation or open reduction with internal fixation within 3 weeks; rehabilitation can take from 3 months to a year.

## 11. CLAVICLE FRACTURE

Imran Shaikh, Robert Cooper



**FIGURE 1.** Displaced clavicle fracture (arrow).

### Clinical Presentation

Direct fall on shoulder or direct trauma to bone are causative.

Midshaft fractures are most common, accounting for approximately 80% of all clavicle fractures.

Thorough neurovascular evaluation is essential, as the clavicle is in close proximity to the great vessels and brachial plexus.

### Diagnosis

Physical exam reveals point tenderness at site; may see visible bone deformity. Shoulder range of motion is limited due to pain.

Best visualized using anteroposterior radiographs; 30° cephalad views may

help confirm diagnosis.

## Management

Treatment is directed toward symptomatic control with splinting, ice, and analgesics.

A sling or figure-of-eight splinting is recommended.

Orthopedic referral is provided in adults with displaced clavicle fractures.

Emergent orthopedic consultation is necessary for open clavicle fractures or neurovascular compromise.

## 12. CLENCHED FIST INJURY

Yanina A. Purim-Shem-Tov



**FIGURE 1.** Infected fight bite injury of the fifth metacarpophalangeal joint.

### Clinical Presentation

Results from a closed fist striking an object (“fight bite” if strikes mouth/teeth)  
Approximately 75% of fight bites result in an injury to bone, tendon, joint, or cartilage from tooth penetration.

Initially, the wound may appear innocuous, but the risk of infection is high.  
When the patient extends the fingers from clenched position, oral bacteria penetrate into deep space.

Presentation is typically delayed (5 to 7 days).

### Diagnosis

Exam and radiographs are usually diagnostic of clenched fist injury. Radiographs will detect fractures, tooth fragments, and, if prolonged infection, possibly osteomyelitis of the bone.

## Management

Appropriate reduction and management of fractures

If presenting without signs of infection, assess and remove foreign objects from the wound; start prophylactic antibiotics to cover oral flora; update tetanus vaccine; 24 to 48 hours follow-up for wound reevaluation

If presenting with signs of infection, consult hand surgery for wound exploration, debridement, and irrigation of joints; start intravenous antibiotics.

## 13. COCCYX FRACTURE

Galetta Carolyn Clayton



**FIGURE 1.** Radiograph of a coccyx fracture.

### Clinical Presentation

Severe, localized pain and tenderness over the coccyx, following traumatic event

Pain exacerbated by sitting and defecation

Follows direct blow to coccyx (tailbone), either from fall onto buttocks or from direct blow in athletic setting

More common in women, as the female pelvis is broader, leaving the coccyx more exposed

### Diagnosis

Diagnosis is established by reproducing symptoms with direct pressure on the coccyx during a rectal examination.

Physical exam should include entire vertebral column, neurologic exam, and rectal exam.



Diagnostic radiographs do not change clinical management. There are high false-negative rates (due to small fractures) and false-positive rates (due to congenital anomalies that may resemble fractures). Exceptions include clinical concern for pathology such as infection or cancer.

## Management

Acute management is generally conservative and supportive, with analgesics, heat or ice, and stool softeners.

Protect the coccyx with “doughnut” or “wedge” cushions to alleviate direct pressure on the coccyx.

Most symptoms resolve over weeks but sometimes over months.

Some patients develop chronic pain (coccydynia), requiring specialist referral.

## 14. DIGIT AMPUTATION

Claire N. Abramoff, Michelle D. Lall



**FIGURE 1.** Clinical photo. (Image courtesy of Menelaos Demestihis, MD.)



**FIGURE 2.** Radiograph. (Image courtesy of Menelaos Demestihis, MD.)

### Clinical Presentation

Results from crush or avulsion injuries or deep finger lacerations  
May bleed significantly and lead to hemorrhagic shock

If reimplantation is possible, wrap the amputated part in saline-soaked gauze, place it in a plastic bag, and then place that in a second plastic bag filled with ice water.

## Diagnosis

History and exam are diagnostic.

X-rays of the injured digit and hand are essential to assess for degree of bony injury and foreign bodies.

## Management

All amputations, except uncomplicated fingertip injuries, require emergency department evaluation by a hand surgeon. The primary initial goal is to evaluate the suitability of the amputated part for reimplantation.

Indications for reimplantation: injuries in children, injuries to the thumb, and multiple digit amputation

Contraindications for reimplantation: crush or avulsion injury, multiple injuries to amputated part, prolonged ischemia time (>24 hours) of amputated part, poor health/significant comorbidities, smoking history

All viable skin should be conserved because it may be needed to provide soft tissue coverage for the amputation stump.

Give prophylactic antibiotics with methicillin-resistant *Staphylococcus aureus* coverage and tetanus booster as needed.

## 15. DISTAL FIBULA FRACTURE

Daniel McCabe

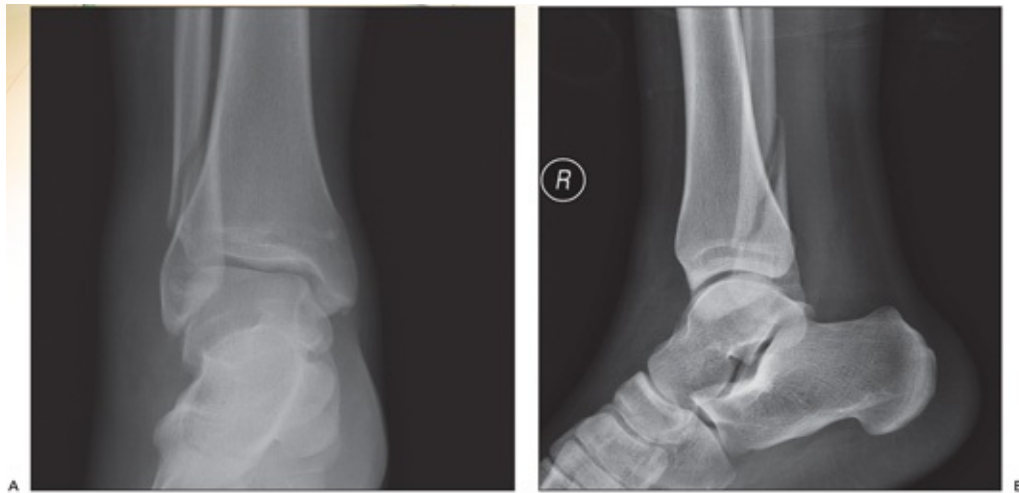


FIGURE 1. Weber B distal fibula fracture. Anteroposterior (A) and lateral (B) radiographs.

### Clinical Presentation

Fracture to fibula at the level of the lateral malleolus

Caused by excessive rotational stress or direct blow to lateral ankle

### Diagnosis

Pain and/or edema over lateral malleolus with x-ray confirmation of fracture

Weber classification: Weber A is below the syndesmosis and associated with transverse fracture. Weber B is at level of syndesmosis associated with a characteristic posterior spike on the distal fragment of the fibula. Weber C is a fracture proximal to the syndesmosis in fibular diaphysis.

### Management

Treatment for isolated lateral malleolar fractures is primarily nonoperative.

Splint the ankle at 90°.

Referral to an orthopedist is recommended when there is suspicion for

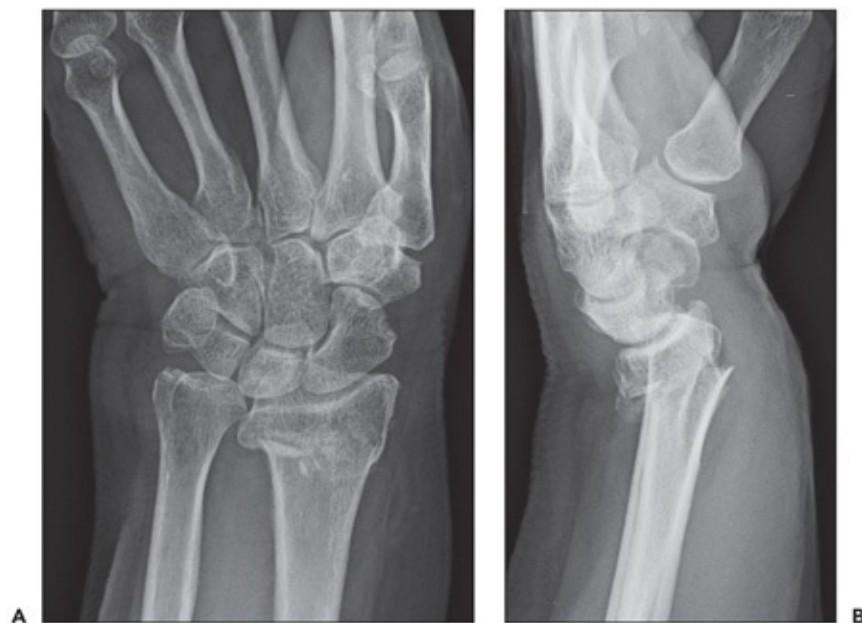
concomitant medial ligamentous injury (medial swelling, ecchymosis, or tenderness), there is >3 mm of fracture displacement, or the fracture is proximal to the ankle joint (Weber B and C).

## 16. DISTAL RADIUS FRACTURE

Michael D. Zwank



**FIGURE 1.** Dinner fork deformity of a Colles fracture.



**FIGURE 2.** Anteroposterior (A) and lateral (B) radiographs.

## Clinical Presentation

One of the most common fractures seen in the emergency department  
Typically caused by fall on outstretched hand mechanism  
Most common is a transverse fracture of the metaphysis with dorsal displacement of the distal fragment (Colles fracture).  
An associated ulnar styloid fracture is often present.  
Open fractures and neurovascular injury are uncommon.

## Diagnosis

Swelling is common; deformity in Colles fractures is referred to as the *dinner fork deformity*.  
Wrist radiographs demonstrate fracture location, displacement, and joint involvement. Measurements of the normal wrist include volar tilt ( $11^\circ$ ), radial length (11 mm), and radial inclination ( $22^\circ$ ). In displaced Colles fractures, there is typically loss of volar tilt, radial length, and radial inclination.

## Management

Goal of reduction is to regain radial length and to achieve some volar tilt of the distal radius fragment.  
Adequate pain control usually achievable with a hematoma block  
Reduction aided by traction using “finger traps”  
Splint with sugar tong being sure to allow adequate finger flexion at metacarpophalangeal joints.  
Close orthopedic follow-up is important, as operative management is common.



## 17. ELBOW DISLOCATION

Lindsey Hogle, Howard Werman, Laura Thompson



**FIGURE 1.** Clinical photo of a posterior elbow dislocation.



**FIGURE 2.** Posterior elbow dislocation with fracture of the coronoid process.

### Clinical Presentation

Elbow dislocation is most commonly posterior.  
Falling onto an outstretched hand is the common mechanism.

The olecranon can be noted and palpated prominently in the posterior direction.

## Diagnosis

Elbow radiographs as well as physical exam aid in the diagnosis.

Prereduction radiographs also assist with identifying associated fractures to the coronoid process or radial head.

Assessment of the neurovascular status of the limb (particularly ulnar nerve) must be completed before and after reduction.

## Management

A commonly used reduction technique involves traction and countertraction. The forearm is placed in the supinated position with the elbow flexed to approximately 90°. Gentle traction is placed on the forearm, whereas countertraction is applied to the humerus with the other hand or by an assistant.

Postreduction x-rays assure appropriate reduction and diagnose any fractures that may have occurred during the reduction.

A posterior splint and sling is applied to the affected arm.

## 18. FELON

Jason P. Stopyra, Adam McHone



FIGURE 1. Clinical photo of a felon.

### Clinical Presentation

The distal fingertip pulp is divided into compartments by 15 to 20 fibrous septae.

A felon is a subcutaneous cellulitis or abscess that develops in the volar distal fingertip.

The thumb and index finger are the most commonly affected digits.

Commonly develops after a puncture wound or may progress from an untreated paronychia

*Staphylococcus* and *Streptococcus* are the most common organisms.

## Diagnosis

Severe tenderness of the volar aspect of the fingertip with fluctuance (abscess) or firm swelling (cellulitis)

Spontaneous purulent discharge and/or an area of pointing may be present.

Plain radiographs should be performed to rule out retained foreign bodies.

## Management

Early pulp infections may be successfully treated with warm soaks and oral antibiotics.

Most pulp space infections require incision and drainage followed by loose packing, splinting, and elevation.

Complications of this procedure include an anesthetic fingertip, digital artery injury, debilitating scar, and mobile finger pad.

Most patients with a felon can be discharged, although patients who are diabetic and/or immunocompromised may need to be admitted.

## 19. FEMORAL NECK FRACTURE

Peter G. Kumasaka



**FIGURE 1.** Right displaced femoral neck fracture.

### Clinical Presentation

Typically an injury of elderly patients, which occurs from a fall from standing  
In younger patient, injury occurs as a result of high-energy mechanism.  
Atraumatic (stress) fracture can occur.  
Some weight bearing possible with nondisplaced femoral neck fractures

### Diagnosis

Affected leg usually externally rotated and shortened.  
Tenderness over hip and pain with axial load or range of motion of hip  
Two-view hip and anteroposterior pelvis x-rays are usually adequate, although  
4% to 8% may be radiographically occult.

If x-ray is nondiagnostic and there is a high index of suspicion, magnetic resonance imaging is best to evaluate for occult fractures.

## Management

Analgesia with parenteral medications or with ultrasound-guided femoral nerve block or fascia iliaca block

Admission and orthopedic consultation

Emergent surgery for young (<60 years of age) if displaced neck fracture because of risk of avascular necrosis

## 20. FEMUR FRACTURE

Stephanie A. Taft



**FIGURE 1.** Spiral fracture of the femur.

### Clinical Presentation

Most often occur in polytrauma patients who have sustained a high-mechanism injury

Patient must also be evaluated for other significant injuries.

If minimal trauma, pathologic fracture should be suspected.

### Diagnosis

Usually obvious rotational and angular deformity of the thigh

Assess neurovascular status as well as all lower extremity joints.

X-rays of the entire femoral shaft as well as x-rays of the hip and knee should



be performed. Concomitant knee injury is present in 20% to 40% of cases. Associated femoral neck fractures are seen in up to 6% of cases but are often missed initially even with hip imaging. Computed tomography may be used to evaluate the femoral neck.

## Management

Traction splinting achieves fracture reduction, pain relief, and control of significant blood loss but should be removed early due to the risk of pressure necrosis and pudendal nerve injury. It should not be used if more proximal or distal injuries of the extremity are suspected.

In addition to intravenous analgesia, a regional femoral nerve block may be considered for pain control.

Traction pinning is used preoperatively until definitive surgical management can be performed.

Surgery (intramedullary nailing) within first 24 hours reduces morbidity and mortality in patients with multiple injuries—timing is dependent on the patient's other injuries and physiologic parameters.

## 21. FINGER DISLOCATION

Imran Shaikh, Robert Cooper



**FIGURE 1.** A lateral PIP joint dislocation of the fifth digit.

### Clinical Presentation

Proximal interphalangeal (PIP) joint dislocations are the most common ligamentous injuries of the hand.

The PIP and distal interphalangeal (DIP) joints dislocate dorsal, lateral, or volar. Dorsal is most common.

Usually sports-related injuries involving high-velocity impact to the end of a finger, causing axial load and hyperextension

### Diagnosis

Physical exam reveals swelling and deformity of the joint. Range of motion is prohibited.

Radiographs help classify the type of dislocation and associated avulsion fractures.

## Management

Digital blocks may be used for anesthesia, although are not always necessary. Reduction is performed by applying longitudinal traction, hyperextension, and direct pressure toward corrected alignment.

The joint should be actively and passively ranged postreduction to assess for ligamentous injury.

The PIP and DIP joints are splinted in extension once reduced.

Any fracture with joint involvement requires an orthopedic referral.

## 22. FINGERTIP AMPUTATION

Scott Bonnono



FIGURE 1. Clinical photo. A: Fingertip. B: Stump.

### Clinical Presentation

The fingertip is defined as distal to the insertions of the flexor and extensor tendons on the distal phalanx.

Fingertip amputation commonly occurs due to accidental trauma, such as crush injury (door) or a sharp instrument (table saw).

### Diagnosis

Diagnosis is based on the history and physical examination.

Physical examination is aided by anesthesia from a digital block and a finger tourniquet to obtain a bloodless field.

Note the structures involved: pulp, nail bed, and bone exposed.

Obtain plain radiographs of finger to help identify extent of bony injury.

### Management

Irrigate the finger extensively with sterile saline.

Assess need for tetanus prophylaxis and stop nonarterial bleeding with pressure.

If bone is exposed, treatment may consist of using a rongeur to trim back the bone so that soft tissue coverage of the tip can be achieved.

Most fingertip amputations can be allowed to heal by secondary intention.

Place a nonadherent (Vaseline) dressing over the tip. If the tip is present and clean, it can be sutured back on to form a “biologic” dressing. Other potential treatments that would require specialty consultation include skin grafts, replantation, and skin/tissue flaps.

## 23. FRACTURE BLISTERS

Bilal Khan, Rashid Kysia



**FIGURE 1.** Fracture blisters of the leg.

### Clinical Presentation

Fracture blisters occur in <3% of fractures requiring hospitalization.

Most common sites of occurrence are areas where thin skin comes in close contact with bone, namely, the ankle, wrist, and elbow. Blisters may develop between 6 and 48 hours after injury.

Shearing forces at the time of the fracture cause injury at the dermal-epidermal junction. Separation between the skin layers is worsened by subsequent edema and venous stasis.

Risk factors predisposing to blister formation include high-energy injuries and the presence of peripheral vascular disease, diabetes, and tobacco use.

## Diagnosis

Patients with recently acquired fractures who complain of pain or pressure should have their splint or cast removed for examination of the underlying skin.

Diagnosis is apparent on visual inspection. Blisters may be hemorrhagic or filled with clear fluid.

Common complications of fracture blisters include infection and ulcer formation.

## Management

There is not yet a consensus as to how these blisters are best managed. As such, the emergency physician should leave the blisters intact and defer management to the orthopedic consultant.

Treatment varies from allowing the injury to resolve spontaneously to early debridement and dressings with silver sulfadiazine.

Operative repair of the underlying fracture and placement of a cast is often delayed until the skin at the blister site reepithelializes, although some argue this is unnecessary.



## 24. GALEAZZI FRACTURE-DISLOCATION

Andrew Yocum, Andrew King



**FIGURE 1.** Note the widening of the DRUJ and radial shortening.

### Clinical Presentation

Isolated fractures of the distal radius with associated subluxation or dislocation of the distal radioulnar joint (DRUJ)

Mechanism is typically a fall with forceful axial loading and forearm torsion.

Can present with forearm compartment syndrome

Anterior interosseous nerve palsy may also be present with paralysis of the flexor pollicis longus and flexor digitorum profundus muscles causing loss of pincer grasp.

### Diagnosis

Pain and soft tissue swelling of the distal third radius and the wrist joint

Plain radiographs with anteroposterior and lateral views of forearm, elbow, and wrist

Radiologic signs suggestive of DRUJ include dorsal or volar radial displacement, ulnar styloid base fracture, widening of DRUJ space, and >5 mm radial shortening.

If unable to appropriately evaluate DRUJ integrity with plain radiographs, computed tomography scan of the forearm is indicated.

## Management

Analgesia with opiate analgesics

Ice application and splinting for comfort

Orthopedic consultation for open reduction and internal fixation due to a high rate of malunion with closed reduction

## 25. GANGLION CYST

Wesley Eilbert, Mark Kirkwood



**FIGURE 1.** Ganglion cyst on the dorsum of the wrist.

### Clinical Presentation

A benign subcutaneous cyst occurring adjacent to joints or tendons

They most commonly occur on the dorsal wrist, although they may occur near any joint of the wrist, hand, or foot.

They are more likely to occur in females and most commonly present in the third to sixth decades of life.

Patients often present with an asymptomatic mass that has been present for months to years, often fluctuating in size.

### Diagnosis

A fluid-filled, rubbery, and slightly mobile subcutaneous mass is palpable.

Average size is 2 to 3 cm in diameter.

Most are painless, but patients may report pain or paresthesias if the cyst places pressure on nerves or adjacent structures.

Diagnosis is usually made clinically. Bedside ultrasound may be used to visualize the cystic structure.

## Management

Patients presenting with uncomplicated ganglion cysts can be reassured that most will resolve spontaneously.

Patients presenting with significant discomfort can be treated conservatively with nonsteroidal antiinflammatory drugs and splinting.

Aspiration of the cyst may provide symptomatic relief, but recurrence is common.

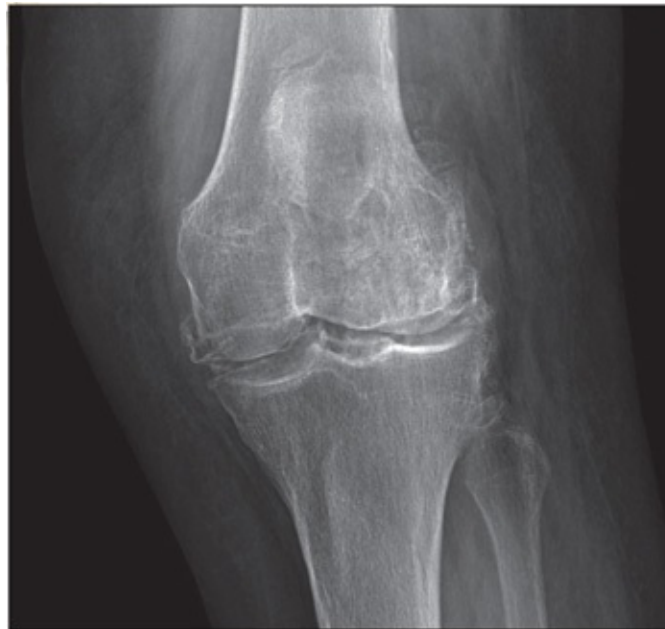
Surgical excision of the cyst may be performed in those patients who fail nonoperative management.

## 26. GOUT AND PSEUDOGOUT

Kim L. Askew



**FIGURE 1.** Erosion of the first metatarsophalangeal joint in gout.



**FIGURE 2.** Chondrocalcinosis of the knee in a patient with pseudogout.

## Clinical Presentation

Arthropathies caused by precipitation of uric acid crystals (gout) and calcium pyrophosphate crystals (pseudogout) in a joint space

Acute onset of edema, pain, and erythema of a single joint; the first metatarsophalangeal joint is frequently affected in gout, whereas the knee is most common in pseudogout; polyarticular involvement in up to 20%.

Risk factors for gout include obesity, hypertension, diabetes, and thiazide diuretic use. Metabolic diseases, such as hyperparathyroidism and hypomagnesemia, precipitate pseudogout.

## Diagnosis

Arthrocentesis is indicated in any patient presenting with new-onset monoarticular arthritis.

Synovial white blood cell count from 10,000 to 70,000/ $\mu$ L and no evidence of bacteria on Gram stain; negatively birefringent, needle-shaped crystals in gout and rhomboid, positively birefringent crystals in pseudogout

Serum uric acid does not predict acute gout presentation or diagnosis. Renal function abnormalities may affect treatment decisions.

Radiographs in gout are typically normal in early disease; however, erosions, tophi, and lytic areas are seen later. Chondrocalcinosis is seen in pseudogout.

## Management

Pain control is typically provided with nonsteroidal antiinflammatory drugs (NSAIDs).

Colchicine is an alternative when NSAIDs are contraindicated (patients with history of peptic ulcer disease and renal insufficiency.) New dosing guidelines exist to reduce toxicity effects. Dosages should be reduced by one-half for patients with a glomerular filtration rate (GFR)  $<50$  mL/min and avoided in patients with hepatic dysfunction, biliary obstruction, or a GFR  $<10$  mL/min.

Corticosteroids are useful for patients when NSAIDs or colchicine are contraindicated. Oral steroids should be tapered over 2 weeks.

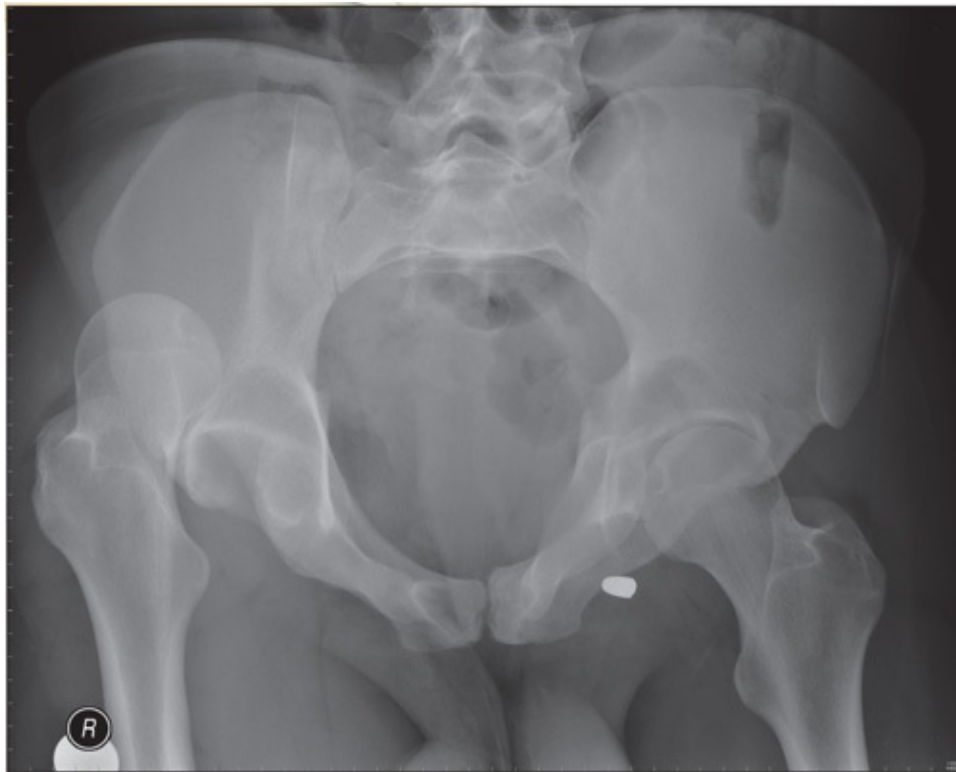
Patients on prophylaxis medications, such as allopurinol, should continue their

regimen.



## 27. HIP DISLOCATION

Panchanan Shukla



**FIGURE 1.** Right posterior hip dislocation.

### Clinical Presentation

Most common type is posterior dislocation, but anterior dislocation can occur. Caused by major force (i.e., high-speed motor vehicle crash) to a flexed knee and hip and can be accompanied by associated fractures to the femur and/or acetabulum

Can occur in the elderly at lower impact

Dislocation can be recurrent in those with a hip prosthesis.

### Diagnosis

Exam shows an internally rotated and shortened lower extremity with

adduction at the hip and an inability to bear weight.

Prereduction imaging to exclude a fracture to the acetabulum, femoral head/shaft, or pelvis

## Management

Reduction achieved through flexing the hip at a 90° angle and applying steady upward force while stabilizing and providing counter traction on pelvis.

Postreduction radiographs for confirmation of successful reduction

Urgent orthopedic consultation is warranted, as the risk of avascular necrosis of the femoral head increases the longer it remains dislocated.

## 28. HUMERAL SHAFT FRACTURE

Michael D. Zwank



**FIGURE 1.** Humeral shaft fracture.

### Clinical Presentation

Presents with pain/swelling/deformity of the upper arm  
Usually results from direct impact to the arm

### Diagnosis

Brachial artery and radial nerve are vulnerable to injury with humeral shaft fractures.

Radial nerve injury causes wrist drop (loss of function of wrist extension).  
Two-view humerus x-rays confirm the diagnosis.

## Management

Treatment is commonly nonsurgical even for significantly displaced fractures. Traction reduction may be used during splinting with goal to attain 1 inch or less of overlapping displacement.

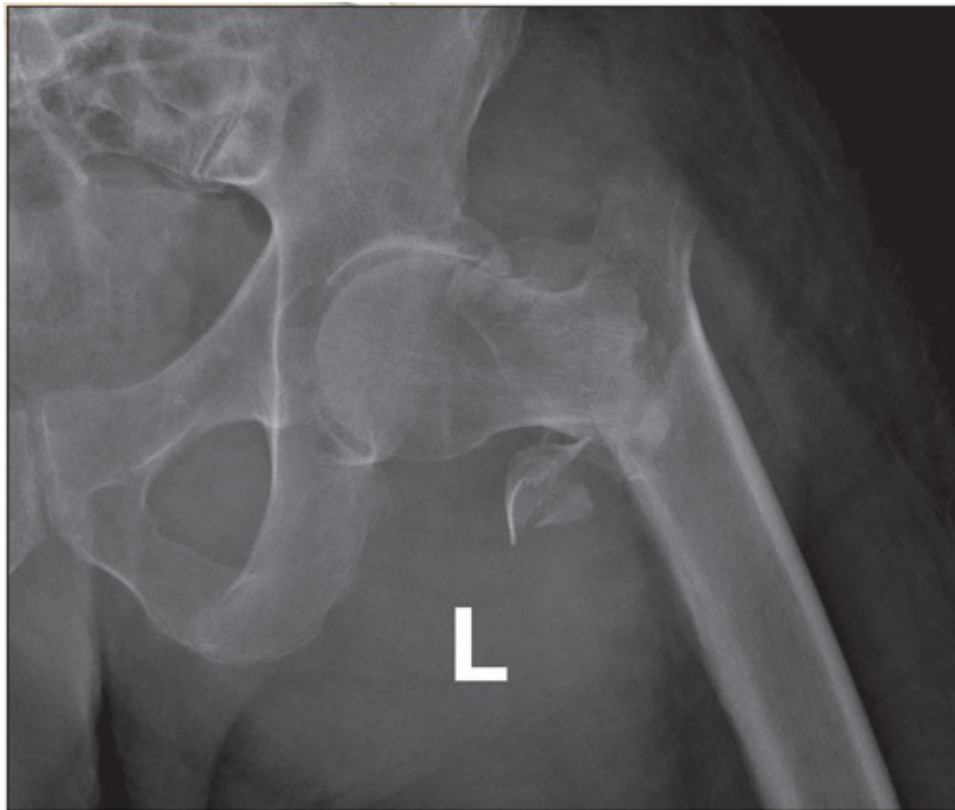
Acute management options include coaptation sugar-tong type wrapping from shoulder around elbow to medial arm or prefabricated shoulder immobilizer or prefabricated humerus cast brace.

Consult orthopedic surgery for any suspected neurovascular injury.

Close orthopedic clinic follow-up is important.

## 29. INTERTROCHANTERIC FRACTURE

Michael Barrie, Andrew King



**FIGURE 1.** Left intertrochanteric hip fracture.

### Clinical Presentation

Intertrochanteric fractures are an extracapsular fracture of the proximal femur between the greater and lesser trochanters.

Suspect in elderly patients with hip pain after a traumatic event.

Patients are unable to bear weight on the affected side; have tenderness to palpation; and pain with external rotation, abduction, or axial loading of the hip.

More obvious fractures will present with the leg in external rotation and shortened.

## Diagnosis

Plain film x-rays of the hip have high diagnostic accuracy; however, occult or x-ray-negative hip fractures make up 3% to 9% of hip fractures.

Magnetic resonance imaging has higher sensitivity compared to computed tomography for detecting hip fractures.

Advanced imaging should be considered in any patient unable to bear weight after a traumatic event.

## Management

Initial management includes evaluating for other injuries including cervical spine injury, early analgesia, and imaging.

Definitive management requires orthopedic consultation and likely operative intervention.

The emergency physician should be aware that delays in surgery have been shown to increase mortality.

Despite definitive surgical management, hip fractures in the elderly have high mortality rate.

## **30. JONES AND PSEUDO-JONES FRACTURES**

David Notley, Andrew King



**FIGURE 1.** Jones fracture.





**FIGURE 2.** Pseudo-Jones fracture.

## Clinical Presentation

Patients will present with pain in the lateral foot and with weight bearing. Jones fracture is located in the metaphyseal–diaphyseal junction. This is in a vascular watershed, and nonunion occurs more frequently.

Pseudo-Jones (Dancer's) fracture is located at the proximal tubercle and occurs due to the pulling of ligaments or tendons on the bony attachments.

## Diagnosis

Tenderness in the lateral foot and resistance to foot eversion is seen on examination.

Radiographs of the foot demonstrate a fracture line that extends into the articulation of the fourth to fifth intermetatarsal in a Jones fracture.

Computed tomography (CT) or magnetic resonance imaging is not routinely used acutely, but CT may be used in follow-up if nonunion is suspected.

## Management

Jones fracture is managed initially with a short leg cast and no weight bearing. Referral to an orthopedic surgeon is imperative, as these are best managed operatively with an intramedullary screw.

Pseudo-Jones fracture is managed nonoperatively in a fracture boot or rigid shoe with weight bearing as tolerated. These injuries are not routinely placed in a cast or splint, as more functional treatment results in better outcomes.

## 31. KIENBÖCK DISEASE

Colette Berube



**FIGURE 1.** Advanced Kienböck disease with lunate collapse.

### Clinical Presentation

Kienböck disease (lunatomalacia) or avascular necrosis of the lunate occurs when blood supply to the lunate is interrupted, causing necrosis and in advanced disease, bony collapse.

The etiology is unknown; however, it is likely a combination of genetic predisposition coupled with repetitive or mechanical stress. The symptoms typically have an insidious onset.

The patient presents with dorsal wrist pain, more frequently in the dominant hand, overlying the lunate, distal to Lister's tubercle on the radius.

Possible factors predisposing patients to the disease are singular blood supply to the lunate, making the bone more vulnerable to compromised vascularity, especially with trauma, and negative ulnar variance (ulna is shorter than the radius), which can cause shear stress on the lunate.

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## Diagnosis

Diagnosis is made radiographically and staged using the Lichtman's classification.

Stage I—Plain films are normal or demonstrate a fracture. Magnetic resonance imaging shows decreased signal on T1- and T2-weighted images.

Stage II—Density changes and sclerosis of the lunate, but carpal bone alignment is maintained.

Stage IIIA—Lunate collapse seen as a shortened and elongated structure in the anteroposterior and sagittal views respectively. Start to see disruption in the carpal bones as the capitate migrates proximally.

Stage IIIB—further disruption in carpal alignment as the scaphoid becomes rotated

Stage IV—arthritis and degeneration of adjacent intercarpal joints

## Management

Treatments for the early stages of disease are wrist immobilization and antiinflammatory medications.

Goals of treatment are to relieve pressure on the lunate in an effort to promote restoration of vascularity.

Later stages of disease require prompt referral to a hand surgeon for possible surgical interventions. Possible treatment options are revascularization, joint leveling of the radius and ulna, or proximal row carpectomy.

## 32. KNEE DISLOCATION

David Calcara, Andrew King



**FIGURE 1.** Anterior knee dislocation.

### Clinical Presentation

Anterior knee dislocation is most common and is due to forced knee hyperextension.

Posterior knee dislocation is the result of a direct tibial force while the knee is flexed. Lateral, medial, and rotational dislocations are least common.

Gross deformity with pain and instability is frequently present. Physical exam may not demonstrate deformity because spontaneous field relocation is common.

Associated with popliteal artery injury in approximately 20% of cases. Assess neurovascular status and obtain ankle brachial indices.

## Diagnosis

Exam is usually diagnostic; do not delay reduction for radiographs if pulses are absent.

Obtain anteroposterior and lateral films to assess for concomitant fracture.

Computed tomography angiography should be strongly considered, as signs and symptoms of arterial damage may be unreliable.

## Management

Perform closed reduction with reassessment of neurovascular status. Flex hip and, while longitudinal traction is applied, hold the tibia and lift the femur into place. Place posterior splint with slight flexion of the knee.

Repair of popliteal artery injuries 8 hours after incident has 80% risk of amputation.

Emergent operation is necessary if there is vascular injury, open dislocation, irreducible dislocation, or compartment syndrome.

### 33. LIPOHEMARTHROSIS

Aaron Ginster, Kristopher Wnek



**FIGURE 1.** Fluid from joint aspiration. Note the fat globules floating on the blood.



**FIGURE 2.** Cross-table lateral radiograph of knee with line demarcating intraarticular fat layer floating on blood layer (arrow).

#### Clinical Presentation

Presence of fat within a hemarthrosis is termed *lipohemarthrosis* and signifies an intraarticular fracture.

Due to release of fat and blood from bone marrow into the joint space

Aids in diagnosis of occult fracture, such as tibial plateau fractures, which are



frequently missed by x-ray

Develops up to 3 hours post-trauma

Occurs in 40% of intraarticular fractures of the knee

## Diagnosis

Grossly visible upon inspection of joint aspirate when pooled in bedside basin;  
fat globules seen floating on the top of the bloody fluid

Best visualized on plain radiographs with a cross-table lateral view  
demonstrating a line separating fat from bloody layer

Also seen on magnetic resonance imaging, computed tomography, and  
ultrasound

## Management

Manage fracture appropriately.

## 34. LISFRANC INJURY

Victor Alcalde, David A. Wald



**FIGURE 1.** AP radiograph demonstrating an avulsion fracture and loss of alignment of middle cuneiform and second metatarsal.



**FIGURE 2.** Radiograph in a second patient with loss of alignment of the second through fifth metatarsals and their midfoot bones.

## Clinical Presentation

Lisfranc injuries are uncommon, accounting for 0.2% of all fractures. These injuries range from sprains to fracture-dislocations.

Lisfranc injuries are seen in motor vehicle accidents resulting from forced hyperplantar flexion of the foot and sporting events where the forefoot is fixed within a foot strap (e.g., equestrian). However, more trivial trauma such as a misstep or a stumble off a curb may also be responsible.

Swelling and tenderness in the midfoot, along with pain and difficulty (or inability) to bear weight

## Diagnosis

Plain radiographs of the foot are usually sufficient for diagnosis. Findings include:

Greater than 2 mm between base of first and second metatarsals on the anteroposterior (AP) view *or* >1-mm difference when compared to the uninjured foot

Fracture of the second metatarsal base (fleck sign) representing an avulsion of

the Lisfranc ligament

Loss of alignment between the metatarsals and cuboids/cuneiforms. Medial borders of 4th metatarsal and cuboid on oblique view. Medial borders of 2nd metatarsal and middle cuneiform on the AP view.

Computed tomography scan may demonstrate small avulsions not seen by plain radiographs. Weight-bearing views and comparison views with the uninjured foot may be helpful when initial radiographs are negative.

## Management

If orthopedic or podiatric consultation is not immediately available, closed reduction can be attempted by hanging the foot by the toes using finger traps and then applying a compressive dressing and a posterior leg splint.

Lisfranc “sprain” is treated by immobilization for 6 weeks.

Lisfranc fracture-dislocation usually requires closed reduction and internal fixation.

Even with appropriate initial treatment, between 40% and 94% of patients develop posttraumatic arthritis.

## 35. LUNATE/PERILUNATE DISLOCATION

Dalia Alwasiyah, Patrick G. Meloy



**FIGURE 1.** Lunate dislocation with spilled tea-cup sign. (Image courtesy of Tarek Hanna, MD.)



**FIGURE 2.** Perilunate dislocation.

## Clinical Presentation

Occurs after high-impact fall on an outstretched hand, with forceful dorsiflexion of the wrist

Patients complain of pain and swelling on the dorsal aspect of the wrist.

On physical exam, there is limited range of motion of the wrist and occasionally associated paresthesia in the median nerve distribution.

## Diagnosis

On plain radiographs, there is overlap of the carpal rows on the anteroposterior view. Lateral radiographs demonstrate loss of the normal alignment of the radius, lunate, and capitate.

Lunate dislocations: spilled teacup sign on the lateral radiograph, in which the lunate is “tipped forward” into the palm. On the posteroanterior view, triangular appearance of the lunate is termed the piece of pie sign.

Perilunate dislocations: Lateral view demonstrates the lunate remaining in position relative to the distal radius while the capitate (and other carpal bones) are dorsally dislocated.

Commonly associated with other injuries, including scaphoid fracture

## Management

Orthopedic consultation in the emergency department (ED)

Reduction in the ED can be attempted with orthopedic support; if successful, immobilization with a long-arm cast is necessary.

Open reduction and internal fixation or arthroscopically guided reduction and pinning are now considered optimal management of these injuries.

## 36. LUXATIO ERECTA

Eddie Markul



**FIGURE 1.** Characteristic raised arm position of luxatio erecta.



**FIGURE 2.** Inferior shoulder dislocation with associated fracture of the greater tuberosity.

### Clinical Presentation

Luxatio erecta, an inferior shoulder dislocation, accounts for <1% of all shoulder dislocations.

Results from forceful hyperabduction of the arm or less commonly by a direct axial load to an abducted arm



The majority of patients will have an associated fracture of the greater tuberosity or a rotator cuff tear.

Greater than 50% of patients will have some degree of neurologic compromise, most commonly an axillary nerve palsy, which typically improves after reduction. Significant vascular compromise has also been documented.

## Diagnosis

Patients present with their arm fixed above their head. Attempts to move the arm will result in significant pain. The humeral head may be palpated along the lateral chest wall.

Plain radiographs demonstrate the humeral head below the glenoid fossa, with the humeral shaft pointed upward.

A thorough neurovascular exam is essential, given the high incidence of associated injuries.

## Management

Closed reduction with procedural sedation will be effective for the majority of cases. Irreducible dislocations occur if the humeral head tears through the inferior capsule and becomes trapped in a “button-hole” defect, necessitating an orthopedic consult for possible open reduction.

Intraarticular glenoid injection of a local anesthetic can improve pain and reduce the amount of medication needed for procedural sedation.

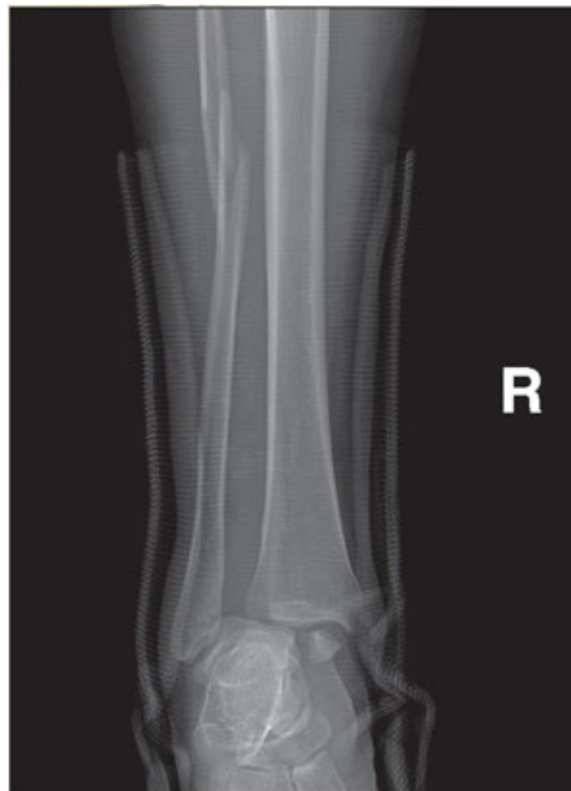
Two techniques have been described for closed reduction: traction-countertraction and the “two-step” method. During the traction-countertraction method, the operator applies axial traction in the direction of the abducted humerus while an assistant applies countertraction by using a sheet placed over the upper trapezius region of the affected shoulder. Traction is maintained as the arm is brought through its arc until it returns to an adducted position at the patient’s waist. During the “two-step” method, the inferior dislocation is first converted into an anterior dislocation before reduction. The operator stands at the head of the patient facing caudally with one hand on the lateral aspect of the midshaft of the humerus and other

positioned over the medial epicondyle. The operator will then push anteriorly on the humerus while stabilizing the medial epicondyle and rotating the arm into adduction. The inferior dislocation has now been converted to an anterior dislocation, which can be reduced using external rotation or the operator's technique of choice.

After reduction, the arm should be placed in a shoulder immobilizer and follow-up arranged with an orthopedic specialist.

## 37. MAISONNEUVE FRACTURE

Henry Z. Pitzele



**FIGURE 1.** Medial malleolar fracture with proximal fibula fracture.

### Clinical Presentation

Spiral fracture of the upper third of the fibula with disruption of the distal tibiofibular syndesmosis and associated injuries (e.g., fracture of the medial malleolus, fracture of the posterior malleolus, or rupture of the deltoid ligament)

Caused by pronation or supination combined with external rotation; most common etiologies are sports; motor vehicle collisions; and slipping on ice, running, and walking.

Patients present with moderate to severe ankle pain and inability to ambulate. Pain or deformity at the proximal fibular fracture site is rare.

Thought to represent 5% of all surgically treated ankle fractures

## Diagnosis

Physical examination of patients with ankle sprain/fracture should include palpation of the proximal fibula.

Any tenderness of the fibula should provoke ordering of plain x-rays of tibia/fibula, with or without weight-bearing stress view of ankle.

Distal neurovascular exam is important, as the peroneal nerve can be injured, causing decreased sensation along top of foot.

## Management

Non-weight-bearing, crutches and a long leg posterior mold

Orthopedic consultation and close follow-up

Most cases require open reduction and internal fixation; prognosis is good for cases promptly diagnosed. Undiagnosed cases risk long-term arthritis and chronic pain.

## 38. Mallet Finger

Michael Gottlieb



FIGURE 1. Clinical photo.



FIGURE 2. Radiograph demonstrating a significant avulsion fracture.

### Clinical Presentation

Results from direct trauma to an extended distal interphalangeal joint (DIPJ), resulting in forced hyperflexion

Tear of the extensor digitorum tendon or avulsion fracture of the proximal aspect of the distal phalanx

Patients will present with the DIPJ in flexion with an inability to extend the distal phalanx.

### Diagnosis

History and examination are usually diagnostic.

Obtain anteroposterior and lateral radiographs of the affected digit to assess for associated fractures, which may require operative management.

## Management

Conservative management includes placing the DIPJ in an extension splint for 6 to 8 weeks.

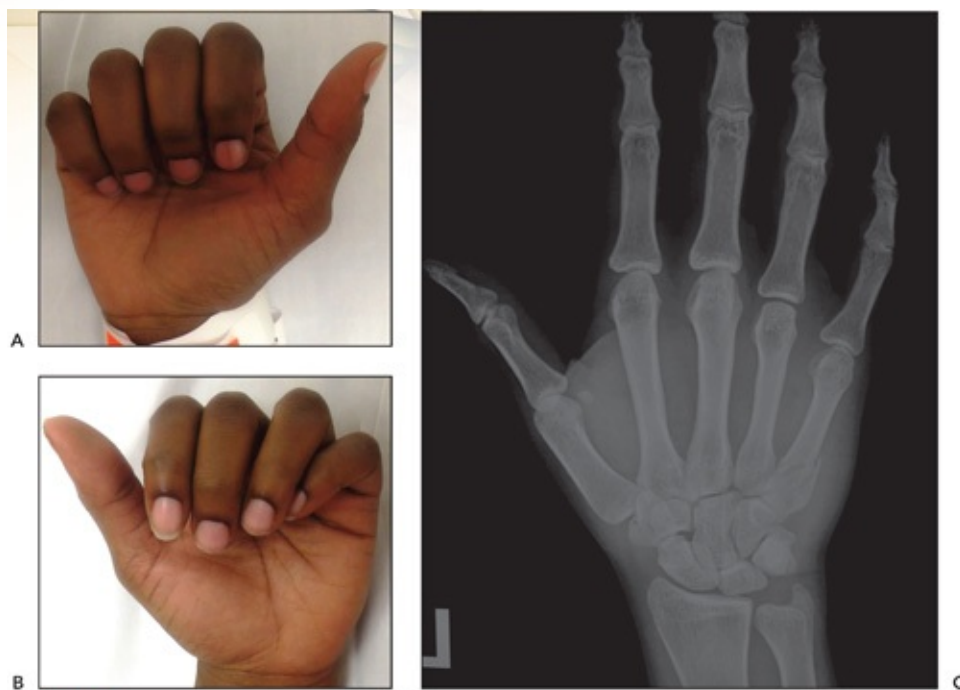
Avoid sustained hyperextension at the DIPJ, as this may lead to ischemia of the skin overlying the dorsum of the DIPJ.

If the DIPJ is flexed at any point during the time period, the healing process must start from the beginning.

Surgery is rarely indicated but may be considered if there is no response to conservative management or if a fracture involves >30% of the articular surface of the DIPJ.

## 39. METACARPAL FRACTURE

Zachariah Ramsey



**FIGURE 1.** A: Normal right hand. B: Left hand with rotational deformity of fifth digit. C: Left hand radiograph of same patient demonstrates nonangulated, nondisplaced fracture at the base of the 5th metacarpal, confirming rotation as the cause of the deformity.

### Clinical Presentation

Metacarpal fractures occur at the base, shaft, neck, or head.

Due to direct blow to the area or a direct axial load

Over a quarter of a million metacarpal fractures in U.S. emergency departments each year; 76% in males

### Diagnosis

Pain, swelling, point tenderness, loss of dorsal metacarpophalangeal (MCP), and possible loss of motion

Test range of motion and perform a neurovascular exam.

Examine for rotational deformity—with all four digits flexed, they should



point toward the scaphoid.  
Radiographs are diagnostic.

## Management

Need adequate analgesia for reduction (hematoma block or regional block)  
Reduction: 90° flexion at affected MCP and proximal interphalangeal joint, then apply upward pressure on middle phalanx and downward pressure over dorsal aspect of fracture

Apply dorsal splint from the interphalangeal (IP) joints to the distal forearm (IP joints in full extension, MCP joint at 70° to 90° of flexion, and wrist with 20° to 30° of extension).

Definitive management is primarily nonoperative. Surgical management may be necessary if any of the following are present: malrotation, shaft angulation >10° to 20° at 2nd/3rd metacarpal or 40° at 4th/5th metacarpal, open or multiple hand fractures, displaced intraarticular fractures.

## 40. METATARSAL FRACTURE

Dalia Alwasiyah, Patrick G. Meloy



**FIGURE 1.** Fractures of the 2nd, 3rd, and 4th metatarsal shafts.

### Clinical Presentation

Classified by anatomic location—shaft, head and neck, and base  
Due to crush injury or from a twisting force with a fixed forefoot  
Patients will have pain, swelling, ecchymosis, difficulty weight bearing, and occasional deformities.

### Diagnosis

On exam, pain is often diffuse and difficult to localize; axial loading of the involved toe is painful.

Three-view x-rays, including posteroanterior, lateral, and oblique, should be

obtained and are usually diagnostic.

Must rule out associated injuries such as phalanx fractures, Lisfranc injury, intraarticular fractures, and metatarsal base fractures

## Management

Nondisplaced fractures of the 2nd to 5th metatarsals can be put in a walking cast and allowed to bear weight as tolerated.

First metatarsal fractures require 4 to 6 weeks of casting and 3 weeks of no weight bearing.

Reduce if displaced  $>3$  mm or angulated  $>10^\circ$ , followed by no weight bearing for 4 to 6 weeks.

Follow-up with orthopedics is required in 2 to 3 days.

Suspected Lisfranc injuries or fractures that are unstable, open, multiple, or have failed closed reduction require orthopedic consultation and surgical repair.

## 41. MONTEGGIA FRACTURE-DISLOCATION

Daniel McCabe

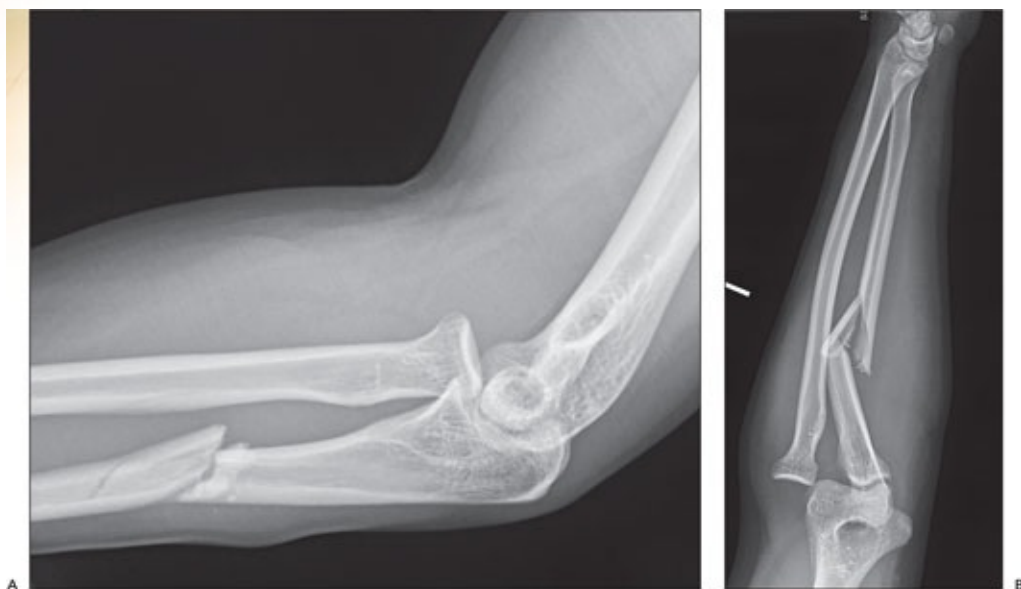


FIGURE 1. Monteggia fracture-dislocations. A: Type I. B: Type III.

### Clinical Presentation

Fracture of proximal third of ulna associated with radial head dislocation  
Due to direct blow or hyperpronation injury  
May be associated with radial or posterior interosseus nerve injury

### Diagnosis

Radiographs are diagnostic; missing the radial head dislocation is prevented by drawing the radiocapitellar line. A line drawn through the shaft of the radius should bisect the capitellum.

Type I (extension type, 60%): anterior fracture angulation and radial head dislocation

Type II (flexion type, 15%): posterior fracture angulation and radial head dislocation

Type III (lateral type, 20%): lateral fracture angulation and radial head

dislocation

Type IV (combined type, 5%): fracture of both ulna and radius with radial head dislocation

## Management

Immobilize the arm in a long arm posterior splint.

Urgent orthopedic consultation and follow-up for reduction and surgical plate placement of the ulna

## 42. NAIL BED INJURY

P. Quincy Moore, David C. Snow



**FIGURE 1.** Nail bed injury.



**FIGURE 2.** After the nail plate is removed, tissue adhesive is used to repair the laceration.

### Clinical Presentation

The nail bed is composed of the epithelial tissue underlying the nail plate.

Usually due to direct blunt trauma or injury by sharp objects such as knives  
Nail bed injuries are often associated with a partial or complete fingertip avulsion.

## Diagnosis

Findings suggestive of nail bed injury include subungual hematoma, bleeding from beneath the nail plate, and fracture or disruption of the nail plate.

Physical exam requires evaluation of sensation with two-point discrimination and vascular integrity by looking for pallor and/or prolonged capillary refill time.

Radiographs should be performed. Distal tuft fractures occur in up to 50% of injuries.

## Management

Subungual hematoma (without nail plate injury) can be trephinated with an 18G needle or electrocautery.

If the nail plate is lacerated or avulsed, it should be removed. Perform a digital block, followed by blunt dissection of the nail from the nail bed.

The 5-0 absorbable sutures or tissue adhesive is used to repair the laceration.

Place a nonadherent gauze or the patient's removed nail back into the nail fold to separate the dorsal roof from the nail bed. Secure with sutures or tissue adhesive.

Prophylactic antibiotics if there is an associated fracture



## 43. NIGHTSTICK FRACTURE

Jason P. Stopyra, Adam McHone



**FIGURE 1.** Nightstick fracture. **A:** Anteroposterior. **B:** Lateral radiographs.

### Clinical Presentation

An ulnar shaft fracture, often due to direct blunt trauma while protecting one's head with the forearm

Historically, this fracture was seen after an assailant resisted arrest from a policeman's nightstick.

More common in men due to a higher participation in contact sports, altercations, falls from heights, and motor vehicle collisions

### Diagnosis

Examine distal neurovascular function and forearm compartments.

Radiographs of the forearm reveal isolated ulnar shaft fracture.  
Radiographs of the elbow and wrist should be considered to exclude additional fractures or fracture-dislocations.

## Management

Nondisplaced or minimally displaced fractures ( $<10^\circ$  of angulation or  $<50\%$  of shaft displacement) may be placed in a splint and sling with orthopedic follow-up.

Displaced or angulated fractures ( $>10^\circ$  of angulation or  $>50\%$  displacement) will likely need rigid plate repair with early range of motion.

Open fractures require emergent consultation and operative management.

Most nightstick fractures heal well with a nonunion rate of only 5%.

## 44. OLECRANON FRACTURE

Asad Nazir



**FIGURE 1.** Displaced olecranon fracture.

### Clinical Presentation

Due to direct trauma or indirectly by forced hyperextension of the elbow during a fall

Exam with tenderness over the olecranon, palpable separation, or inability to extend the elbow against resistance

The ulnar nerve is most vulnerable to injury, resulting in decreased sensation of fifth digit and hypothenar eminence or motor weakness of interossei muscles.

### Diagnosis

Lateral radiographs offer the best view.

The degree of comminution, extent of articular surface disruption, and amount of displacement in the 90° flexion position should be assessed.

In adolescents, the diagnosis can be aided by comparison films and the presence of an abnormal fat pad.

## Management

Olecranon fractures should be immobilized with the elbow in flexion at 45° to 90° using a long arm posterior splint.

Ice, elevation, analgesics, and referral to an orthopedist

Displacement >2 mm requires surgical repair.

The presence of ulnar nerve symptoms should prompt orthopedic consultation.

## 45. OPEN FRACTURE

Sierra Beck



FIGURE 1. Type IIIB open fracture.

### Clinical Presentation

Maintain high clinical suspicion for open fractures and perform a thorough exam, as wounds may be subtle.

Any wound near a fracture site should be assumed to communicate with the fracture and be treated as an open fracture until proven otherwise.

Open fractures do not always require high-energy mechanism.

### Diagnosis

Gustilo classification system: I—clean wound that is  $<1$  cm; II— $>1$  cm wound without extensive soft tissue damage; III-extensive soft tissue laceration ( $>10$  cm) and further classified as follows; IIIA—soft tissue loss;

IIIB—extensive bone damage, periosteal stripping, or large contamination requiring reconstruction; IIIC—associated vascular injury requiring repair

## Management

Copious irrigation as early as possible, then cover with sterile saline–moistened or sterile petroleum–impregnated gauze

Prompt administration of antibiotics. Type I fractures should receive intravenous first-generation cephalosporin. Types II and III fractures require additional gram-negative coverage (i.e., aminoglycoside). Wounds that are grossly contaminated with soil or farm material should have penicillin added to cover *Clostridium*.

Tetanus vaccine should be updated where appropriate.

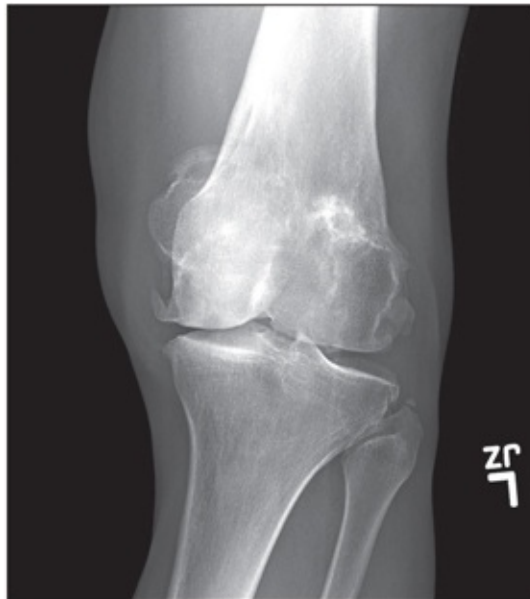
Early orthopedic consultation

## 46. OSTEOARTHRITIS

Daniel McCabe



**FIGURE 1.** Osteoarthritis of hand.



**FIGURE 2.** Radiograph of knee osteoarthritis.



## Clinical Presentation

Due to disruption between balance of synthesis and degradation of extracellular matrix

Primary (idiopathic) versus secondary (posttraumatic, metabolic disorders, inflammatory, gout)

Leading cause of disability of individuals age >65 years

Risk factors: age, obesity, injury, repetitive stress on joints

## Diagnosis

Slowly developing joint pain with stiffness worse with movement and relieved with rest

At late stage of disease, there can be decreased range of motion and crepitus.

No systemic findings (localized to joint), minimal joint inflammation

Weight-bearing joints and distal interphalangeal/proximal interphalangeal (PIP) joints commonly affected; distinguished from rheumatoid arthritis, which spares PIP joints

Radiographs with uneven distribution of decreased joint space and occasionally osteophytes

## Management

Weight reduction in overweight individuals, acetaminophen, nonsteroidal antiinflammatory drugs

Corticosteroids do not change course of disease.

Aerobic exercises improve long-term outcome, but patient should refrain from overuse.

Overweight individuals can change course with weight loss.

## 47. PARONYCHIA, ACUTE

Paul Casey



FIGURE 1. Clinical photo of a paronychia.

### Clinical Presentation

Most common infection of the hand

*Staphylococcus aureus* is most common causative bacteria.

Associated with nail biting, trauma from manicure, or foreign bodies

### Diagnosis

Diagnosis is clinical; based on tenderness, erythema, and swelling along lateral nail fold due to pus accumulation  
Must be distinguished from herpetic whitlow

## Management

Consider digital block. Incision and drainage using a no. 11 blade scalpel to gently elevate the eponychial fold from the nail, releasing pus from the paronychia cavity

Antibiotics if extensive surrounding cellulitis, immune compromise, diabetes, or peripheral vascular disease

Tetanus prophylaxis

## 48. Patella Dislocation

Scott C. Sherman

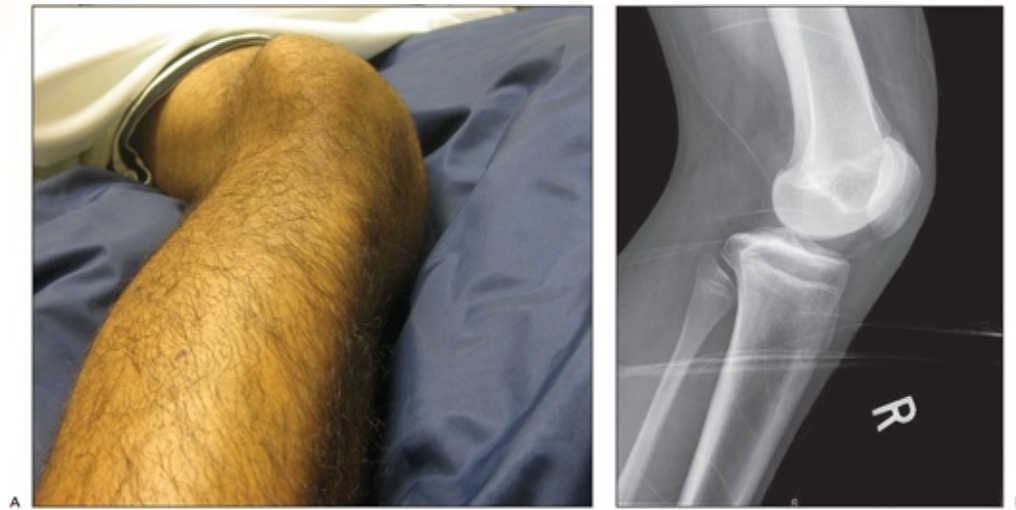


FIGURE 1. Patella dislocation. A: Clinical photograph. B: Radiograph.

### Clinical Presentation

The most common location for patella dislocation is lateral.

Dislocation is frequently recurrent.

Direct trauma to the patella with the knee flexed or an indirect mechanism (quadriceps contraction with knee flexion) are causative.

### Diagnosis

Exam and radiographs are usually diagnostic if the patella remains dislocated. Prereduction radiographs exclude a fracture.

Spontaneous reduction is detected by the *patellar apprehension test*. The knee is flexed to 30° and the patella is pushed laterally. If the patient feels the sensation of impending redislocation, the test is positive.

### Management

Reduction is achieved by flexing the hip initially.

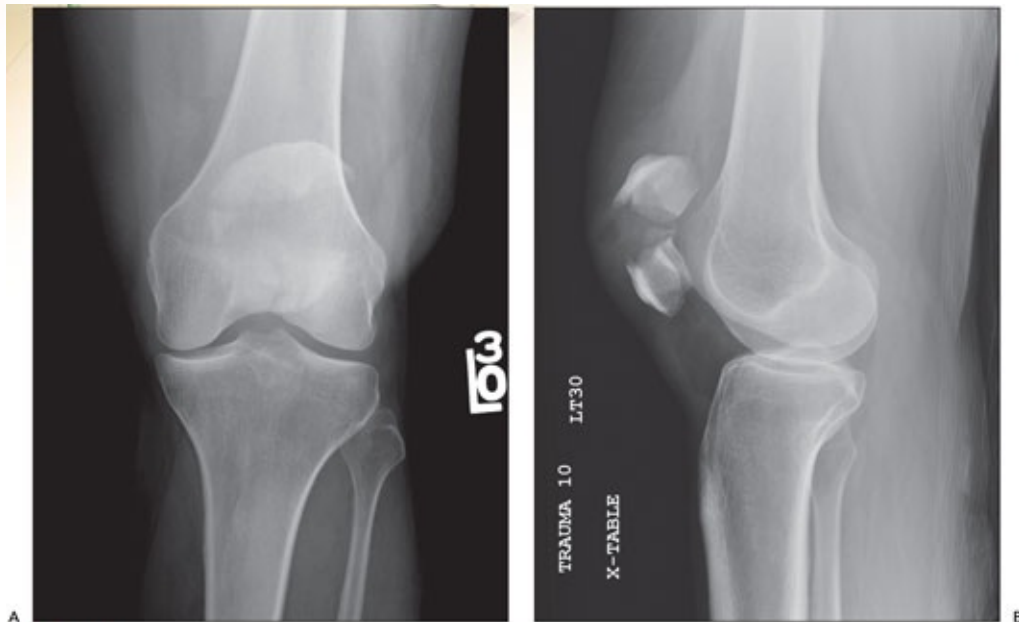
While extending the knee, gentle pressure is applied over the patella in a medial direction.

Postreduction radiographs can be obtained to determine the success of reduction.

A knee immobilizer is placed, keeping the knee in full extension.

## 49. PATELLA FRACTURE

Natasha Demehri, Michelle D. Lall



**FIGURE 1.** Transverse patella fracture with displacement that will require surgery. **A:** Anteroposterior. **B:** Lateral views. (Images courtesy of Tarek Hanna, MD.)

### Clinical Presentation

Acutely swollen knee with ecchymosis, joint effusion, and patellar tenderness secondary to direct or indirect knee trauma

Transverse fracture results from a forceful contracture of the quadriceps tendon, often occurring in younger adults. There is inability to actively extend the knee.

Stellate or comminuted fracture usually results from direct forces, for example, falling directly onto the knee or striking the knee against a dashboard in a motor vehicle collision

### Diagnosis

Anteroposterior, lateral, and sunrise views; lateral view is best to assess displacement. Sunrise view helps to assess osteochondral fractures and

vertical fractures.

Bipartite and multipartite patellas are normal anatomic variants and should not be confused with fractures.

## Management

Patella fractures are treated with a knee immobilizer, crutches, and orthopedic referral.

Nondisplaced fracture with intact extensor function does not require surgery; partial weight bearing, as tolerated

Fractures with  $>2$  mm displacement,  $>3$  mm of fragment separation, comminuted fractures with displacement, and those with interruption of the extensor mechanism will require operative repair. These patients should maintain non-weight-bearing status.

Any patient with an open fracture should have orthopedic consultation in the emergency department.



## 50. PATELLAR TENDON RUPTURE

Sean P. Boley, Samuel J. Stelpflug



**FIGURE 1.** Note the patella's superior position in patellar tendon rupture.



**FIGURE 2.** Lateral radiograph demonstrating patella alta.

## Clinical Presentation

The patient typically presents with an acutely painful knee and an inability to actively extend the knee or bear weight.

Patellar tendon rupture tends to occur in individuals younger than 40 years of age.

Injury often occurs in the setting of trauma, especially with eccentric contraction and violent jumping activities.

## Diagnosis

On exam, there is inability to actively extend the knee. There is a palpable defect in the tendon, although this finding is limited due to the swelling and hematoma formation. Additionally, the patella is palpated in an abnormally superior position (patella alta).

In the case of complete rupture of the patellar tendon, plain radiographs are diagnostic with the visualization of patella alta. Advanced imaging (magnetic resonance imaging) is indicated only if the diagnosis is suspected in the absence of obvious patella alta.

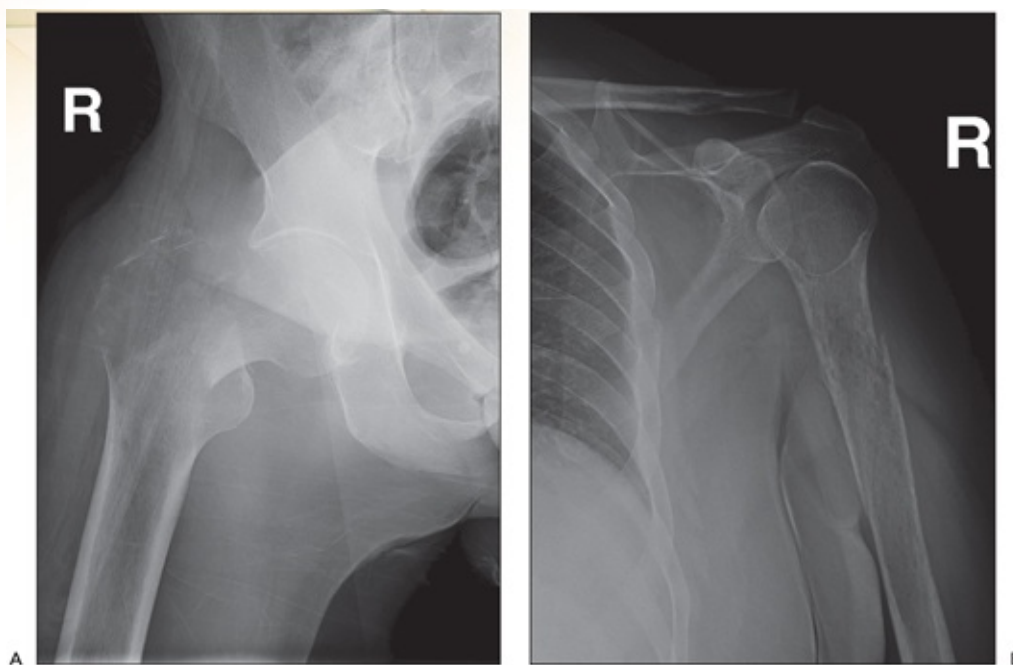
## Management

Prompt referral to an orthopedic surgeon should be made as soon as the diagnosis is made, as early operative fixation (within 7 to 10 days) is crucial. Prior to fixation, the knee should be kept in a knee immobilizer, and patients should be instructed to weight bear as tolerated.

Early operative fixation predicts very good clinical outcome (range of motion and muscle strength) with few complications. Late repair is associated with rerupture of the tendon as well as knee stiffness.

## 51. PATHOLOGIC FRACTURE

Jessica Folk



**FIGURE 1.** Pathologic fractures. **A:** Femoral neck. **B:** Humerus.

### Clinical Presentation

A fracture of weakened bone following relatively minor trauma  
Most often secondary to malignancy  
Involving the axial skeleton and proximal limbs  
Patients present with localized pain seemingly out of proportion given the mechanism.

### Diagnosis

Plain films show loss of trabecular structure and distinct borders.  
Lesions appear osteolytic or “moth-eaten.”  
Axial injuries typically result in vertebral body collapse.  
Radiographs are usually sufficient for initial imaging.

Magnetic resonance imaging or computed tomography with intravenous (IV) contrast further demarcates bone marrow and soft tissue involvement.

## Management

Pain control with IV analgesics

Bone metastases usually require chronic pain management.

Palliative radiation improves pain in the majority of patients.

Orthopedic or neurosurgical consult, as many require open surgical repair

## 52. PELVIS FRACTURE

Michael D. Zwank



**FIGURE 1.** Right superior and inferior pubic rami fractures (arrows).

### Clinical Presentation

Pelvis bones form a “ring” that can be fractured in many locations depending on degree and location of force.

Usually requires high-energy mechanism such as motor vehicle crash and pedestrian struck

Important to differentiate stable from unstable fractures—often based on location and severity

Involvement of sacroiliac joint/posterior elements generally indicates unstable fracture.

## Diagnosis

Diagnosis made by anteroposterior pelvis x-ray and assisted by inlet/outlet pelvis x-rays or by computed tomography (CT) scan

CT scan is modality of choice in patients with high suspicion and negative x-rays or in patients with severe trauma.

Evaluate patient carefully for other traumatic injuries, as these are common.

## Management

Stable pelvis fractures can be treated with simple analgesic medication.

Open book–type injuries with widening of pubic symphysis can be stabilized with pelvic wrapping.

Wrapping may have role in other pelvis fractures based on clinical judgment.

Unstable fractures are often associated with severe hemorrhage and may need large transfusions.

Arterial angiography plays an important role in the definitive care of severe pelvis fractures.

## 53. PILON FRACTURE

John Cook



**FIGURE 1.** Pilon fracture with characteristic intraarticular comminuted pattern.

### Clinical Presentation

Due to a high-energy axial load of talus into distal tibia caused by a motor vehicle collision or fall from height

Characterized by distal tibial comminution and significant soft tissue swelling

Associated injuries include calcaneal, tibial plateau, pelvic, and vertebral compression fractures.

An open fracture in 20% to 25% of cases



## Diagnosis

Anteroposterior, lateral, and mortise views of the ankle should be obtained as well as tibia/fibula and foot films.

Computed tomography scan for surgical planning for repair of articular fragments

## Management

Orthopedic consultation in the emergency department; elevate leg and monitor for compartment syndrome.

Due to a high incidence of soft tissue complications, meticulous examination of skin for breaks and skin blisters is required, as these fractures can convert from closed to open.

Pilon fractures require operative repair. Delayed fixation is common to allow appropriate wound care and for soft tissue swelling to decrease.

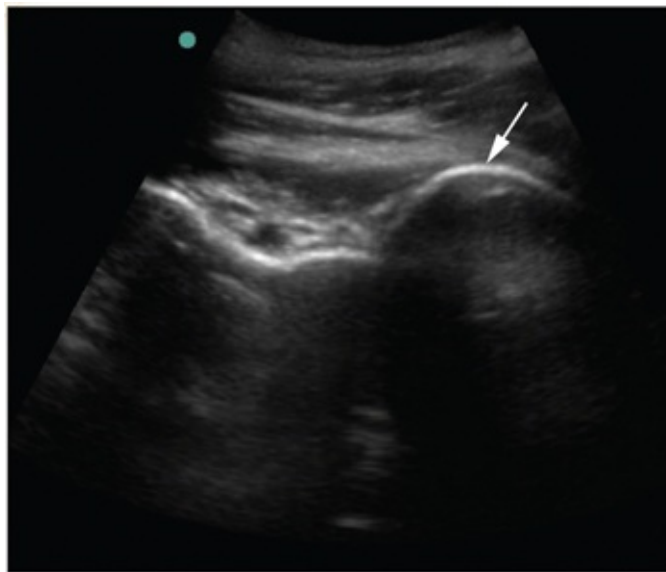
Nonoperative management with a long leg cast is reserved for nondisplaced fractures or patients who are severely debilitated.

## 54. POSTERIOR SHOULDER DISLOCATION

Sierra Beck



**FIGURE 1.** Light bulb and trough line signs.



**FIGURE 2.** Ultrasound demonstrating a posteriorly dislocated humeral head (arrow).

## Clinical Presentation

Posterior shoulder dislocations represent <3% of all shoulder dislocations. Posterior dislocations are classically described in association with seizures or electrocution but most commonly occur from direct trauma to the anterior shoulder.

The arm is held adducted and in internal rotation. Anteriorly, the coracoid process may appear prominent. The humeral head may be palpated posteriorly. The patient will be unable to externally rotate the shoulder.

## Diagnosis

Radiographic changes are often subtle or inconclusive. Diagnosis is missed in 50% of cases.

Classically, anteroposterior shoulder radiographs are described as having a “light bulb” sign where the humeral head takes on a more circular appearance in internal rotation. A “rim sign” occurs, as the humeral head is displaced laterally by the posterior glenoid rim and the normal half-moon overlap between the glenoid and humeral head is lost. The “trough line” sign refers to a line visualized within the humeral head that corresponds to the base of the impaction fracture produced by the posterior glenoid.

Scapular Y and axillary views aid in the diagnosis.

Bedside ultrasound; the low-frequency curvilinear ultrasound probe is placed over the posterior shoulder parallel to the scapular spine. In a normal shoulder, the humeral head articulates with the glenoid. In posterior dislocation, the humeral head will be seen in the near field of the image and it will not articulate with the joint when passive external rotation is performed.

## Management

Reduction is performed with downward traction and slow external rotation of the shoulder as pressure is applied on the posterior shoulder over the dislocated humeral head.

Reduction can be difficult to perform and often requires procedural sedation. Following reduction, full range of motion and external rotation of the shoulder

should be present. The shoulder should be immobilized and orthopedic follow-up arranged.

## 55. PREPATELLAR BURSITIS

Panchanan Shukla



**FIGURE 1.** A large noninfected prepatellar bursitis.

### Clinical Presentation

Inflammatory swelling of the knee bursa between the patella and overlying skin

Commonly caused by direct pressure from repetitive kneeling or by trauma to the knee such as from a fall

Presents with pain and swelling over the front of the knee

On occasion can become infected, most commonly by *Staphylococcus aureus*

### Diagnosis

Exam demonstrates tenderness over bursal sac and cystic fluid collection palpable above the patella.

Range of motion of the knee is not affected, and this can help distinguish it from an acute knee effusion.

Radiography may show signs of soft tissue swelling but is usually nondiagnostic, and diagnosis based largely on history and clinical exam.

## Management

Knee padding and/or brace will help provide compression and reduce swelling.

Nonsteroidal antiinflammatory drugs to reduce pain and swelling

If clinically warranted, bursal aspiration and drainage may be necessary for therapeutic and diagnostic purposes to rule out septic bursitis.

## 56. PROXIMAL HUMERUS FRACTURE

Michael D. Zwank



FIGURE 1. Surgical neck fracture.

### Clinical Presentation

Predominantly an injury of older individuals (mean age 63 years old)  
Caused by direct trauma or fall on outstretched arm  
Multiple locations of fracture follow old epiphyseal planes including anatomic neck, surgical neck, and greater/lesser tuberosity.  
Arm of patient typically held close to body with limited movement

### Diagnosis

Three-view shoulder x-ray makes diagnosis and fracture pattern clear.



May also consider two-view humerus x-ray if humerus shaft fracture is suspected

Fracture often minimally displaced

## Management

If associated glenohumeral dislocation, emergent operative management is usually necessary.

If significantly displaced, consider reduction and orthopedic consultation. Sling or prefabricated shoulder immobilizer is usually adequate for acute management.

Adhesive capsulitis is risk if prolonged immobilization.

Early orthopedic clinic follow-up is important.

## 57. QUADRICEPS TENDON RUPTURE

Panchanan Shukla



**FIGURE 1.** Defect noted superior to the right patella.



**FIGURE 2.** Superior pole avulsion is frequently seen on lateral radiograph.

### Clinical Presentation

Tendonitis, steroid use, fluoroquinolones, and chronic diseases that disrupt

blood flow can weaken the tendon and make it more likely to tear.  
Often occurs when there is heavy load on the leg with foot planted and knee partially bent  
Presents as a tearing or popping sensation followed by pain and swelling to the knee

## Diagnosis

Tenderness with a possible indentation superior to the patella  
Decreased ability to fully extend the knee and/or inability to do a straight leg raise  
Radiographs demonstrate patella baja (inferior displaced patella) and sometimes an avulsion of bone from the superior portion of the patella.  
Ultrasound will show an anechoic region at the rupture site; however, magnetic resonance imaging is the test of choice to confirm and characterize extent of tear.

## Management

Knee immobilizer and urgent orthopedic consultation  
Small tears can be treated nonoperatively with physical therapy if knee extension is minimally affected.  
Complete tears are treated with surgery, typically a few days to a week after the injury.

## 58. RADIAL HEAD FRACTURES

Paul Bobryshev, Trevor Lewis



FIGURE 1. Radial head fracture (arrow).

### Clinical Presentation

Radial head fractures are the most common fractures of the elbow in adults. Due to a fall onto an outstretched hand causing the radial head to be driven into the capitellum

Mason classification: nondisplaced (type I), displaced  $>2$  mm (type II), comminuted (type III), associated elbow dislocation (type IV)

### Diagnosis

Tenderness over the radial head and pain with pronation and supination of the forearm

Obtain anteroposterior, lateral, and oblique views; often best seen on oblique views

Subtle or occult fracture identification can be aided by looking for elevation of the anterior and posterior fat pads on the lateral radiograph.

Greenspan view (modified lateral view obtained by angling tube 45° toward the radial head) or computed tomography scanning if clinical suspicion for fracture is high with normal x-rays

## Management

Ice, analgesics, and referral to orthopedic surgeon within 1 week

Nondisplaced fractures with no mobility restriction can be treated with sling.

Displaced fractures require a posterior long arm splint.

Comminuted fractures require operative intervention, often with radial head resection and prosthetic replacement.

Associated elbow dislocations must be reduced prior to discharge.

## 59. RHEUMATOID ARTHRITIS

Chuang-yuan Lin



**FIGURE 1.** Hands of a patient with chronically untreated rheumatoid arthritis.

### Clinical Presentation

Rheumatoid arthritis (RA) is a chronic, progressive, waxing/waning polyarticular symmetric joint disease.

Peripheral joint involvement is more common, notably in fingers, wrist, hand, and midfoot.

Axial skeletal involvement becomes an issue in chronic RA disease.

Ulnar deviation of wrist and fingers and boutonnière deformity in chronic disease

### Diagnosis

RA is a clinical diagnosis. In emergency department setting, consider serum

rheumatoid factor as a screening tool (sensitive but not as specific). Rule out septic joint disease if the patient presents with only poly- or oligoarticular pain with fever, erythema, and/or warmth overlying the affected joint.

## Management

Anticipate a “difficult” airway if the RA patient requires intubation—it may be difficult to adequately position the head and neck.

Primary goals of RA flares are pain management and close follow-up with primary care provider or rheumatologist.

Nonsteroidal antiinflammatory drugs are front line for pain control. A steroid burst may help for the short term. Chronic steroid suppressive therapy may be required—but do so only in conjunction with a rheumatologist.

Disease-modifying antirheumatic drugs are effective but associated with gastrointestinal, hepatic, and immunologic complications.



## 60. SCAPHOID FRACTURE

Sorabh Khandelwal, Leigh Ann Giano



**FIGURE 1.** Scaphoid view demonstrating fracture of the scaphoid waist.

### Clinical Presentation

Most common carpal bone to be fractured (60% to 70% of all carpal fractures)  
Fall on outstretched hand with forceful hyperextension of wrist or direct axial compression

Associated injuries including radius fracture, another carpal bone fracture, or carpal dislocation in up to 10% of cases

Scaphoid (especially the proximal portion of the bone) has a tenuous blood supply, making fractures at risk for avascular necrosis, delayed union, or nonunion, which can lead to early degenerative arthritis.

## Diagnosis

Focal tenderness is dependent on site of fracture: (1) volar prominence at the distal wrist crease for distal pole fractures, (2) anatomic snuffbox for waist fractures, and (3) just distal to Lister's tubercle for proximal pole fractures. Standard wrist radiographs (posteroanterior [PA], lateral, and oblique) miss up to 20% of scaphoid fractures. Scaphoid view (ulnar deviated PA) may aid in diagnosis.

Computed tomography has improved sensitivity, but magnetic resonance imaging is considered the gold standard for definitive diagnosis.

## Management

Fractures should be placed in a thumb spica splint with orthopedic follow-up. Clinically suspected fractures with normal radiographs should be treated as fractures; they should be splinted with repeat exam and imaging in 7 to 10 days.

Scaphoid fractures with >1 mm displacement, angulation, or comminution need surgical repair.

## 61. SCAPHOLUNATE DISSOCIATION

Dalia Alwasiyah, Michelle D. Lall



**FIGURE 1.** Note the diastasis between the scaphoid and lunate (arrow).

### Clinical Presentation

Fall on an outstretched hand with impact on the thenar eminence or forceful extension of the wrist

Pain or clicking sensation over the radial aspect of the wrist

Recurrent visits for chronic wrist pain should raise the suspicion of undiagnosed dissociation.

### Diagnosis

Applying pressure with your thumb on the scaphoid will elicit pain, and the scaphoid may be unstable. Watson's shift test may produce a clunk or snap. Posteroanterior (PA) radiograph view demonstrates a scapholunate gap  $>3$  mm, classically known as the *Terry Thomas Sign*.

Routine radiographs may appear normal, so additional stress views are

recommended. These include clenched fist and ulnar-deviated anteroposterior views, which will accentuate widening of the scapholunate joint.

The rotation of the scaphoid as it tilts toward the observer on a PA view will enhance the cortex producing a *cortical ring sign*.

On the lateral view, the scapholunate angle will be  $>60^\circ$ .

## Management

Thumb spica or radial gutter splint

Urgent orthopedic consultation is necessary, as patients generally require surgical reduction.

Even properly recognized and treated, patients may have chronic arthritis and wrist instability.

## 62. SCAPULAR WINGING

Henry Z. Pitzele



**FIGURE 1.** Patient with scapular winging.

### Clinical Presentation

Results from damage to the long thoracic nerve or C5–C7 nerve roots causing paralysis of serratus anterior muscle

Etiologies include trauma, compression, neuropathy, and iatrogenic damage during surgery.

Patients present with discomfort and weakness of upper extremity.

### Diagnosis

On examination, the medial border of the scapula is asymmetrically prominent or elevated.

Winging is worsened with forward flexion of arm and with pushing (i.e.,

pushing against a wall).

Anterior arm flexion is limited to 90° and abduction to 110°.

Radiographs of cervical spine, chest, and shoulder to rule out mimics

## Management

Pain control including sling if necessary; referral to an orthopedist for follow-up

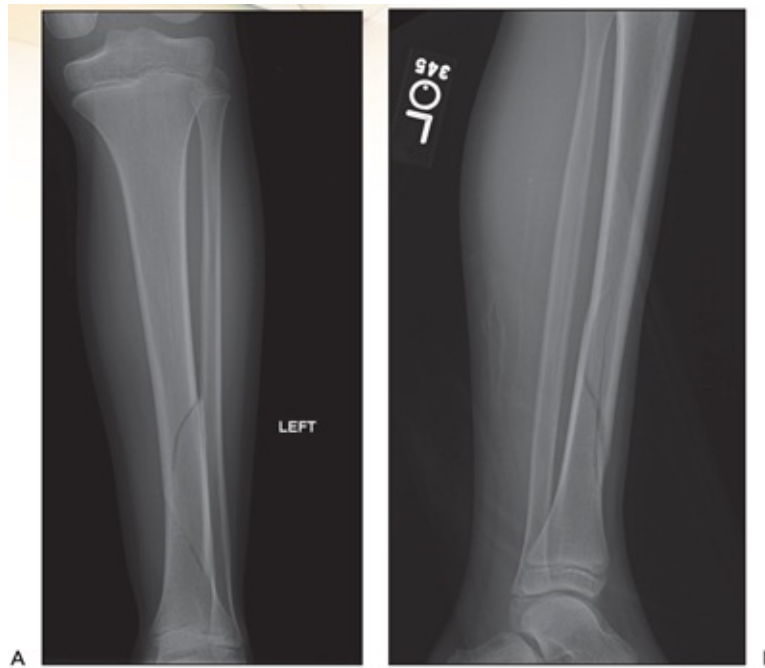
Approximately 75% of cases functionally resolve with minimal deficits through conservative therapy within 24 months.

Conservative therapy includes pain control, range of motion exercises, and occasionally a specialized “winger’s brace.”

Surgical correction by orthopedist required if no improvement with conservative therapy.

## 63. SPIRAL FRACTURE

Claire N. Abramoff, Michelle D. Lall



**FIGURE 1.** Spiral fracture of the distal tibia. **A:** Anteroposterior. **B:** Lateral. (Images courtesy of F. Joseph Simeone, MD.)

### Clinical Presentation

Due to a torsional or twisting force

The most common spiral fracture, *toddler's fracture*, is a nondisplaced fracture of the distal tibia. It is now referred to as the *childhood accidental spiral tibial* (CAST) fracture and occurs in children up to 8 years old.

Spiral fractures of the humerus and femur in nonambulatory children are rare, and nonaccidental trauma or pathologic fractures should be highly considered.

### Diagnosis

Patients with humeral spiral fractures may only have tenderness or mild edema at the site of the injury. Thorough examination to test for radial nerve



injury is required.

Patients with a CAST fracture may have a generally normal exam but will likely refuse to ambulate or bear weight on the affected side.

Distal tibial fractures are difficult to image; anterior, posterior, and oblique imaging is required to adequately visualize the fracture line.

If concerned for nonaccidental trauma or pathologic fracture, the patient may need an extensive workup as an inpatient.

## Management

CAST fractures require a long-leg splint, and the patient should have orthopedic follow-up within 1 week. If radiographs are not conclusive, a splint should be applied and repeat x-rays can be obtained at the patient's follow-up visit.

Humeral spiral fractures have excellent healing due to a strong periosteum, and a sling and swathe is the only needed treatment.

Pediatric femoral spiral fractures are very rare, and orthopedics should be consulted for management.

Displaced spiral fractures require orthopedic consultation because they will often require surgical management.

## 64. SPONDYLOLISTHESIS

Damali Nakitende



**FIGURE 1.** Grade 1 spondylolisthesis of L4 on L5.

### Clinical Presentation

Spondylolisthesis is defined as a disruption of the pars interarticularis causing the superior vertebra to shift forward.

Disruption of the pars interarticularis can be due to trauma and degenerative changes such as osteoarthritis, pathologic (malignancy), or congenital malformation of the joint facets.

The most common presenting symptom is lower back pain that may radiate to the buttocks or posterior thighs. Pain is worsened by ambulation, rotational movements, and hyperextension.

Spondylolisthesis can be graded according to degree of slippage of the superior vertebra in relationship with the caudal one (grade 1: <25% slippage, grade II: 26% to 50%, grade III: 51% to 75%, and grade IV: 76% to 100%).

Patient with high grades of spondylolisthesis may develop a variety of neurologic symptoms associated with the back pain due to the compression of nerve roots.

## Diagnosis

Guided by a combination of the history, physical exam, and imaging  
Physical exam will reveal pain with examination of the back and neurologic deficits in more severe cases.

Plain films and computed tomography can be used as the initial screening tools.

Magnetic resonance imaging is the most sensitive imaging modality to make diagnosis and is indicated in patient with neurologic symptoms.

## Management

Low-grade spondylolisthesis can be managed with pain control and physical therapy. Surgery may be indicated in cases where pain is refractory to conservative management or neurologic deficits are noted.

High-grade spondylolisthesis management is age-dependent. Children typically have surgical stabilization due to risk of further slippage. Adults are usually managed conservatively with pain control and physical therapy before surgery is considered.

## 65. STERNOCLAVICULAR JOINT DISLOCATION

Daniel McCabe, George Chiampas



**FIGURE 1.** Posterior left sternoclavicular dislocation. **A:** Notice the prominent swelling at medial clavicle; **B:** CT reconstruction. (Reprinted from Beecroft M, Sherman SC. Posterior displacement of a proximal epiphyseal clavicle fracture. *J Emerg Med.* 2007;33(3):245–248. Copyright 2007, with permission from Elsevier.)

### Clinical Presentation

Clavicle articulates the sternum with a saddle-type joint; stabilized by costoclavicular and sternoclavicular ligaments

Due to direct, high-energy force to medial clavicle or compression of shoulder girdle medially with the shoulder thrust forward

Medial clavicle can dislocate anteriorly or posteriorly relative to manubrium.

Posterior dislocation can cause internal injury such as tracheal compression or erosion, blood vessel compression with congestion, mediastinal injuries, compression of esophagus, or pneumothorax.

Very rare injury accounting for <1% of all dislocations; anterior is more common than posterior dislocation.

Secondary injuries can be present due to large force required for this injury to occur. Provider should evaluate for other injuries such as pneumothorax, hemothorax, and rib fractures.

### Diagnosis

Swelling at medial end of clavicle with anterior dislocation. With a posterior dislocation, localized swelling can make it difficult to detect a depression of

the clavicle clinically. With either type, there is pain at the superior chest and shoulder with any shoulder movement or with supine positioning. Computed tomography (CT) with intravenous contrast provides the diagnosis and evaluation of surrounding structures. Plain radiographs can be attained with cephalic angle although not sensitive enough to reliably diagnose injury.

## Management

Anterior dislocation: No immediate treatment needed. Referral for urgent reduction by orthopedic surgeon within 24 hours should be arranged.

Reduction technique: Put patient in supine position, place folded towel behind shoulders, abduct arm, apply lateral traction, and push medial clavicle into place.

Posterior dislocation: orthopedic and thoracic surgery consult. Reduction required immediately if airway compromised. Cardiovascular surgeon needed if vascular structures are compromised or hematoma noted on CT. Patient will need urgent reduction (<24 hours). Reduction technique: Put patient in supine position, place folded towel between shoulders, abduct arm, apply lateral traction, and pull medial clavicle anteriorly with fingers or towel clip. If closed reduction is unsuccessful, then surgical repair is indicated.

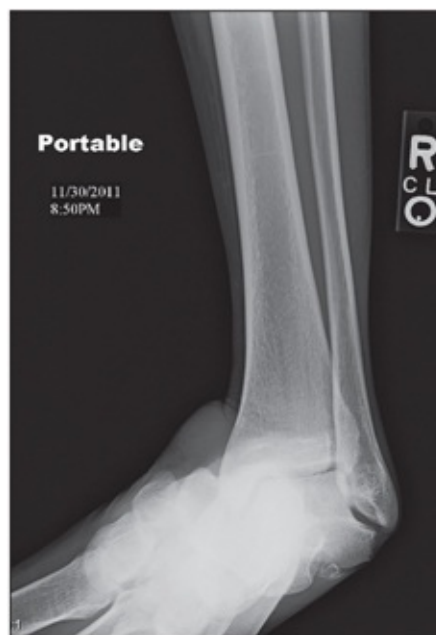
Patient should be placed in figure-of-eight harness with orthopedic follow-up.

## 66. SUBTALAR DISLOCATION

John Cook



**FIGURE 1.** Medial subtalar dislocation of the left foot.



**FIGURE 2.** Radiograph of subtalar dislocation.



## Clinical Presentation

Subtalar dislocation is a rare injury defined by the simultaneous disruption of the talocalcaneal and talonavicular joints while the ankle mortise remains intact.

Due to landing on an inverted or everted foot, leading to a medial (85%) or lateral (15%) subtalar dislocation, respectively

Ten percent of subtalar dislocations present as an open fracture-dislocation, and 50% have associated fractures.

## Diagnosis

Medial dislocation presents with foot and calcaneus medially, the head of the talus dorsolateral, and the foot in supination and plantar flexion.

Lateral dislocation presents with calcaneus and foot lateral to the talus, the head of the talus lies medially, and the foot appears in pronation.

Ankle and midfoot radiographs, with the foot anteroposterior view usually being most valuable.

## Management

Majority of cases can be managed with procedural sedation and closed reduction in the emergency department.

Closed reduction is facilitated by knee flexion to relax the gastrocnemius.

Longitudinal distraction of the foot often accompanied by an initial exaggeration of the mechanism to “release” the calcaneus, followed by reversal of the deformity leads to reduction; a clunk may be appreciated with successful reduction.

Successful reduction should be followed by splinting with a posterior short-leg and U-shaped splint with the ankle put in about 90° of dorsiflexion.

Reduction can be blocked by the talonavicular joint capsule, the extensor digitorum brevis tendon, or the posterior tibialis tendon and may necessitate operative management.

The subtalar joint is usually stable after reduction but often requires a period of immobilization for 4 to 6 weeks with close orthopedic follow-up.



## 67. SUBUNGUAL HEMATOMA

Nathan Finnerty, Andrew King



FIGURE 1. Subungual hematoma.



FIGURE 2. Following trephination.

### Clinical Presentation

Accumulation of blood under the nail without fracture of the nail plate

Most commonly from a crush injury

Patients typically present with throbbing pain secondary to increased pressure beneath the nail.

## Diagnosis

Clinical diagnosis based on mechanism of injury and physical examination findings

Radiographs should be performed to evaluate for fractures, most commonly tuft fractures.

Careful examination of the nail margin and fold, as disruption will alter management

## Management

Trephination of the nail plate for decompression and drainage of the hematoma

Trephination may be performed with 18G needle, scalpel, or electrocautery.

Electrocautery is fast and relatively painless; however, flammables and acrylic nails should be removed prior to procedure. Other methods will likely require digital nerve block.

After drainage, provide general wound care instructions. Soak the affected finger in warm, soapy water two to three times daily for 7 days. Fractures should be splinted with support of the distal joint in extension.

Disruption of the nail margin or fold requires nail plate removal for nail bed repair.

## 68. TENDON LACERATION

Stephen Park



FIGURE 1. Extensor tendon laceration of the thumb.

### Clinical Presentation

Due to a sharp object, which lacerates skin and underlying flexor or extensor tendon

Injury of tendon classified into zones depending on location. Flexor tendon injury has five zones. The Kleinert classification is used for extensor tendons and is divided into eight zones from the distal interphalangeal (DIP) joint to proximal to the wrist. Zone I is over the DIP joint. Zone II is over the middle phalanx. Joints are odd-numbered zones and even are over bones.

It is important to determine if the injury is caused by human teeth, also known as *fight bite*.

### Diagnosis

Careful physical exam and wound exploration after hemostasis has been obtained is key to diagnosis.

Radiographs may be necessary to rule out fracture or retained foreign body. Test both active range of motion and strength against resistance of digits in isolation.

Examine tendon in full range of motion of finger during wound exploration. Neurovascular exam important with flexor tendon injury, as tendon runs close to digital neurovascular bundles

## Management

Irrigate wound, close skin loosely with simple interrupted sutures (unless dirty wound or fight bite), prescribe antibiotics, and refer to hand surgeon ideally within 7 days of injury.

Flexor tendon injuries: Splint with metacarpophalangeal (MCP) joint at 50° flexion and interphalangeal joints 20° flexion.

Extensor tendon injury: zone I and II injury: dorsal splint holding DIP in extension. Zone III to VIII: volar splint with MCPs in extension and wrist extension from 20° to 45°.

Partial tendon injury may require surgical repair if >50% width of injury.

## 69. TIBIAL PLATEAU FRACTURE

Michael Gottlieb



**FIGURE 1.** Split depression fracture of the lateral tibial plateau.

### Clinical Presentation

The tibial plateau is the most proximal portion of the tibial bone and is a critical weight-bearing area for the lower extremities.

Fractures occur in a bimodal distribution, occurring with high-energy trauma in younger patients and low-energy trauma in older and osteoporotic individuals.

Due to valgus or varus force applied to an axially loaded knee, such as when a car bumper strikes a pedestrian's knee

Presents with a large joint effusion, tenderness along the joint line, and an

inability to bear weight

## Diagnosis

Standard knee radiographs (anteroposterior, lateral, and oblique view) may miss subtle fractures.

Computed tomography is more sensitive and better able to fully delineate the injury.

Lipohemarthrosis (on x-ray or arthrocentesis) is highly suggestive of an intraarticular fracture.

## Management

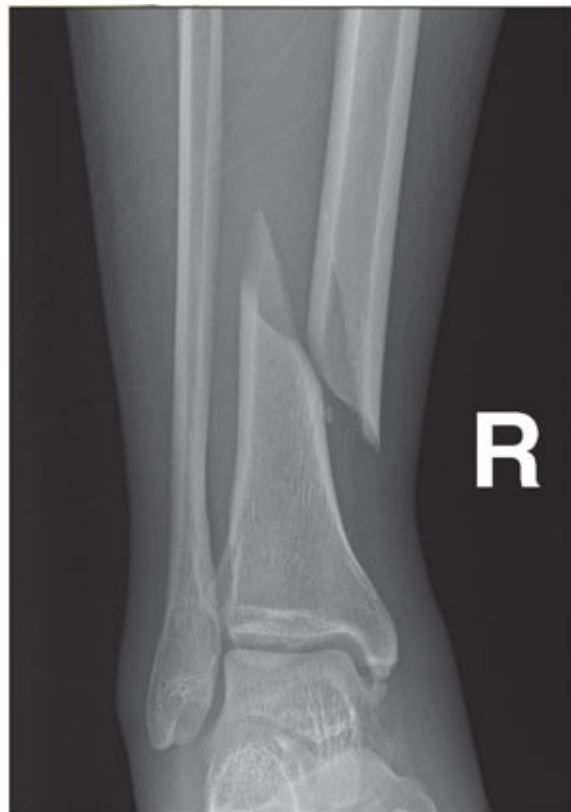
Long leg posterior mold splint, crutches, and referral to an orthopedic surgeon

Many patients will require surgical fixation.

Missed fractures are associated with risk of decreased mobility, joint instability, and posttraumatic arthritis.

## 70. TIBIAL SHAFT FRACTURE

Elena Strunk, Michelle Sergel



**FIGURE 1.** Displaced spiral fracture of the tibial shaft.

### Clinical Presentation

The most commonly fractured long bone in the body  
Open fractures and compartment syndrome are potential complications.  
Isolated tibial shaft fractures are rare and typically the fibula is involved.  
Transverse shaft fractures are due to a direct blow to the bone. Spiral fractures are due to rotational forces. A comminuted fracture pattern is usually a result of a high-energy impact injury.

### Diagnosis



Pain and inability to bear weight on the injured leg

Exam and radiographs are usually diagnostic. Full-length anteroposterior (AP) and lateral views of the affected tibia, along with lateral and AP views of the ipsilateral knee and ankle, should be obtained.

## Management

Administer analgesics.

Examine the compartments and skin for an open fracture and check neurovascular status.

Long leg posterior splint in order to avoid further soft tissue injury

The subsequent management of tibial shaft fractures depends on alignment and whether the fracture is closed or open. Most patients require surgery.

Nonoperative management is reserved for patients with closed low-energy fractures with acceptable alignment (50% or more of cortical contact,  $<10^{\circ}$  to  $15^{\circ}$  of angulation on lateral film,  $<10^{\circ}$  of angulation on AP film,  $<5^{\circ}$  of rotation deformity).

## 71. TOE DISLOCATION

Galeta Carolyn Clayton



**FIGURE 1.** Open toe dislocation.



**FIGURE 2.** Radiograph of a toe dislocation.

### Clinical Presentation

Direct trauma to the toe and/or foot  
Presents with pain and often obvious deformity  
Most dislocations are dorsal; may be associated with a fracture

## Diagnosis

Exam should include site of maximal pain and swelling, neurovascular status, presence of wounds, and range of motion of involved toe.  
Radiographs should be obtained to confirm dislocation and to rule out associated fracture.

## Management

Perform a digital block. Reduce a dorsal dislocation by having an assistant brace the patient's foot. Gently hyperextend the joint while pushing the base of the dislocated phalanx into place.  
Following reduction, test joint stability through the full range of motion of the digit, obtain a postreduction plain radiograph, buddy tape the affected toe to its adjacent toe for 2 weeks, and refer for outpatient follow-up.  
Consider orthopedic or podiatry consultation for open or irreducible dislocation, neurovascular compromise, interphalangeal joint dislocation of the great toe, or multiple metatarsophalangeal dislocations.

## 72. TOE FRACTURE

Brendan Devine



**FIGURE 1.** Proximal phalanx fractures. Intraarticular fracture of great toe and shaft fracture of third toe (arrows).

### Clinical Presentation

Phalanx fractures are the most common fracture of the forefoot, with fractures of the proximal phalanx of the great toe being the most frequently encountered.

Direct trauma from a falling object, axial loading (“stubbed toe”), or abduction forces

Pain, swelling, ecchymosis, and deformity of the toe within the first several hours; occasionally, subungual hematomas may also be noted.

## Diagnosis

Anteroposterior and oblique plain films; lateral views are less helpful due to bony overlap.

## Management

Nondisplaced or minimally displaced fractures of the lesser toes can be treated with dynamic splinting and hard-soled shoe, typically for a period of 2 to 3 weeks.

Displaced fractures should be reduced with a digital block and traction.

Fractures of the great toe involving >25% of the joint space should be referred to orthopedic specialist and managed either with dynamic splinting or posterior splinting with hard-soled shoe.

Subungual hematomas should be drained with electrocautery or 18G needle.

## 73. TRIGGER FINGER

Elana Tan



**FIGURE 1.** Trigger finger of the 4th digit.

### Clinical Presentation

Repetitive use causes inflammation and scarring leading to nodule formation on the flexor tendon sheath at the metacarpophalangeal joint.

The nodule prevents smooth sliding of the tendon over the A1 pulley at the base of the digit.

Finger or thumb may “lock” or “catch” in flexion and straighten with a “pop,” like releasing a trigger on a gun.

More common in diabetics

### Diagnosis

Based on history of the finger “locking” into flexion and physical examination  
Local tenderness and/or swelling at volar base of the digit  
Pain elicited with extension, often accompanied by a “popping” sensation when the finger or thumb is straightened  
Radiographs are not necessary to make diagnosis.

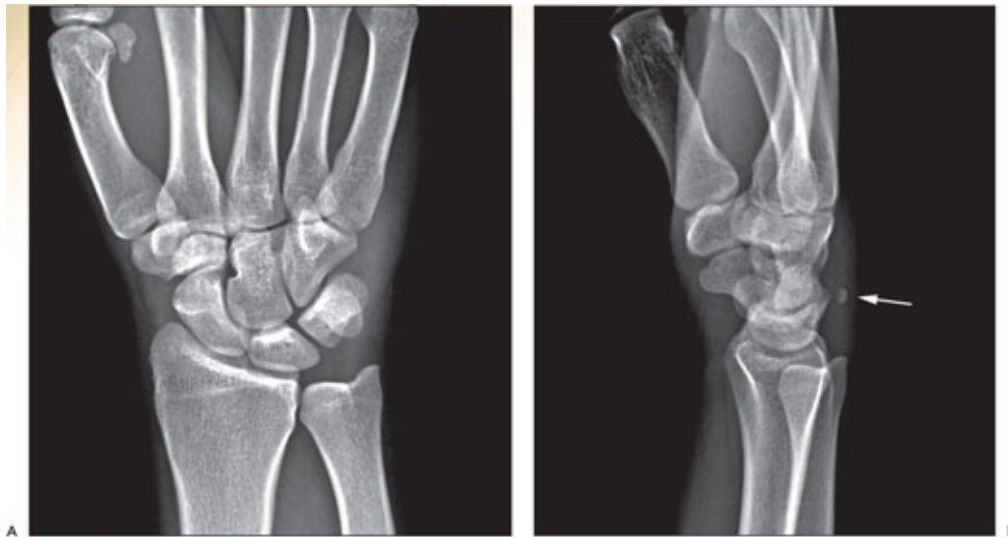
## Management

Conservative treatment includes rest, ice, antiinflammatory medications, and buddy tape or a finger splint to immobilize the affected digit.  
Glucocorticoid injection into the tendon sheath for persistent symptoms  
Surgical release of the A1 pulley ligament is usually curative for symptoms refractory to conservative treatment and steroid injection.



## 74. TRIQUETRAL FRACTURE

Thomas Adam Criswell



**FIGURE 1.** Avulsion fracture is seen on the lateral projection only. **A:** Anteroposterior. **B:** Lateral (arrow).

### Clinical Presentation

Second most common carpal bone fracture after scaphoid  
Mechanism typically is fall on dorsiflexed and ulnar deviated wrist.  
Greater than 90% are avulsion or “chip” fracture of dorsal cortex.  
Body fractures are generally caused by direct blow and frequently associated with perilunate dislocation or other injuries.

### Diagnosis

Point tenderness over dorsal ulnar aspect of wrist  
Posteroanterior, lateral, and oblique wrist radiographs  
Avulsion fracture is seen on dorsal side of lateral projection.

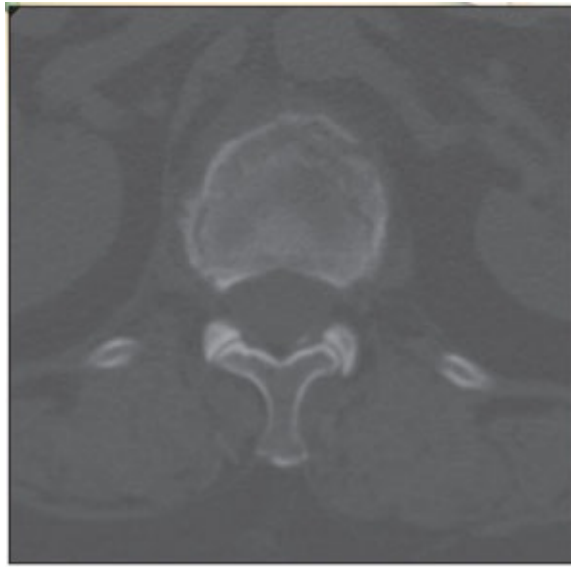
### Management

Volar splint from proximal forearm to the midpalm.

Avulsion fractures should remain immobilized 3 to 4 weeks until asymptomatic; surgery rarely required and only if pain persists. Body fractures require cast immobilization for 4 to 6 weeks if minimally displaced or surgical fixation for those with associated ligamentous injury or significant displacement.

## 75. VERTEBRAL COMPRESSION FRACTURE

Timothy J. Rittenberry, Mark Kirkwood



**FIGURE 1.** Vertebral compression fracture on CT.



**FIGURE 2.** L1 Vertebral compression fracture.

### Clinical Presentation

Vertebral compression fractures (VCFs) are due to collapse of a vertebral body

with axial loading forces.

Most commonly associated with osteoporosis in the geriatric population.

Decreased bone mineral density predisposes these patients to VCF.

May also occur due to lytic bone lesions secondary to malignancy

VCF can occur in severe osteoporosis or malignancy with seemingly trivial mechanisms of injury (e.g., sneezing, stepping off of a curb, heavy lifting).

High-energy trauma is usually needed to cause VCF in otherwise healthy patients. They can be seen in falls from extreme heights, motor vehicle collisions, or extreme contact sports.

Although some patients will present complaining of back pain, many older patients with VCF may be asymptomatic.

Patients may have midline spinal tenderness upon palpation, most often at lower thoracic or upper lumbar levels.

Decreased stature and increased lumbar lordosis are often noted in older patients with VCF.

## Diagnosis

A thorough history should be obtained, with special attention to mechanism of injury and symptom onset.

Lateral spine plain radiography will show decreased vertebral height, most often in the anterior segment of vertebral body. VCF may also be visible on computed tomography (CT) scan.

Magnetic resonance imaging may be necessary if there is concern of secondary intervertebral or neurologic injury.

## Management

Most VCFs are stable and may be managed conservatively.

Associated neurologic impairment is rare.

Patients with unstable compression fractures or those not responding to conservative therapy may need elective surgical interventions such as kyphoplasty or vertebroplasty.

Patients presenting to the emergency department with significant discomfort should be treated symptomatically. Use caution when treating elderly with

nonsteroidal antiinflammatory drugs.

All patients should have follow-up with their primary care provider, and if appropriate, an orthopedic/spine specialist.



SECTION

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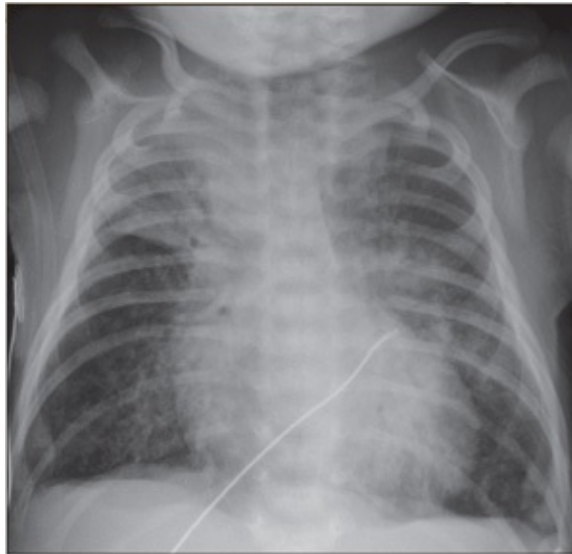
PEDIATRICS

SECTION EDITOR

Stephen John Cico

# 1. BRONCHIOLITIS

Marisa C. Louie



**FIGURE 1.** Bronchiolitis x-ray with significant atelectasis.



**FIGURE 2.** Bronchiolitis x-ray.

## Clinical Presentation

Bronchiolitis is the most common lower respiratory infection in children younger than 2 years of age and is a leading cause of infant hospitalizations.



Although respiratory syncytial virus (RSV) is the best described etiology, many viruses can cause bronchiolitis including rhinovirus, human metapneumovirus, influenza, and parainfluenza.

Patients develop upper respiratory tract symptoms such as rhinorrhea followed in a few days by lower respiratory symptoms including retractions, wheezing, and/or crackles. The very young may present with apnea.

Long-term immunity to RSV does not develop, so patients may develop bronchiolitis multiple times.

## Diagnosis

Diagnosis is entirely clinical, based on the history and physical exam. Chest radiographs should not be routinely obtained but can be considered if the diagnosis is uncertain or in cases of severe disease. Streaky perihilar infiltrates are often identified. Atelectasis can be present to varying degrees and often leads to a misdiagnosis of pneumonia.

Viral testing for RSV does not usually change management but might be useful in cases of diagnostic uncertainty.

When fever is present, evaluation for serious bacterial infection should occur in keeping with the provider's usual practice with respect to age; however, such infections are uncommon in patients with bronchiolitis.

## Management

Treatment is entirely supportive. Increased work of breathing and insensible losses may result in dehydration, and mild hypoxemia often occurs. Local practice varies regarding when supplemental oxygen is needed; the American Academy of Pediatrics recommends a threshold of 90% for otherwise healthy infants at sea level.

Most children with mild to moderate disease do not require hospitalization but should receive close follow-up. Risk factors for severe disease include age less than 12 weeks, prematurity, congenital heart or chronic lung disease, and immunosuppression.

Corticosteroids have no role in the treatment of bronchiolitis.

Bronchodilators may improve the exam and work of breathing in a minority of

patients. However, bronchodilators do not affect the duration of illness or need for hospitalization.

Recurrent bronchiolitis can be difficult to distinguish from reactive airway disease or asthma. Standard asthma therapies might benefit patients with recurrent wheezing and atopy or strong family history of asthma.

## 2. CANDIDAL DIAPER DERMATITIS

Michele M. Walsh, Danielle Mercurio



FIGURE 1. Irritant candidal dermatitis.



FIGURE 2. Candidal dermatitis.

### Clinical Presentation

Generally starts with 3 to 4 days of diaper dermatitis

May occur in setting of recent diarrheal illness or in 6- to 24-month-olds who drink bottles during the night

Macerated, inflamed skin is contacted by genital and gastrointestinal tract

flora.

After  $\geq 72$  hours of contact, a secondary skin infection with *Candida albicans* occurs.

## Diagnosis

Candidal dermatitis consists of clusters of papules and pustules, coalescing into well-demarcated beefy red rash on the genitals, thigh creases, and perineum.

Erythematous oval satellite lesions occur among the periphery of the rash and may extend onto thighs or up toward the umbilicus.

Other considerations include diaper dermatitis, perianal cellulitis (group A *Streptococcus*), atopic dermatitis, and rare conditions, such as Langerhans cell histiocytosis, congenital immunodeficiencies, and neonatal HIV.

## Management

Topical antifungal medications such as nystatin or imidazole ointments with every diaper change until skin is well-healed

In addition, standard treatment of irritant diaper dermatitis is paramount to healing and includes frequent diaper changes, gently and thoroughly cleansing affected skin with alcohol-free wipes and applying barriers ointments.

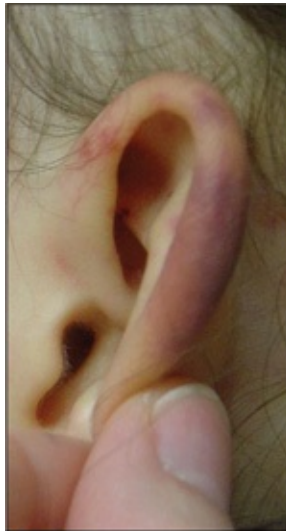
By avoiding contact with moisture, further skin inflammation and breakdown will be prevented, allowing skin to heal.

### 3. CHILD ABUSE—PHYSICAL

Stephen John Cico



**FIGURE 1.** Classic metaphyseal fracture of distal tibia.



**FIGURE 2.** Ear bruising consistent with nonaccidental trauma.



**FIGURE 3.** Excessive bruising in nonaccidental trauma.

## Clinical Presentation

Over 700,000 substantiated cases of child abuse and neglect occur in the United States every year.

It is the leading cause of trauma-related death in children younger than the age of 4 years.

Abuse must always be considered in children with unusual or unexplained injuries or who have delayed presentations.

Inconsistent or changing histories should be well-documented in the medical record.

## Diagnosis

Unusual bruising in unusual number or pattern, particularly on the trunk/neck/ears in children younger than 4 years of age, should raise a suspicion of abuse. Any bruising in preambulatory children are also concerning for abuse.

Spiral fractures in preambulatory children, metaphyseal fractures, posterior rib fractures, and fractures in multiple stages of healing

Head computed tomography should be performed as part of the workup in children younger than 1 year of age, and skeletal surveys should be performed in children younger than 2 years of age.

## Management

These patients need to be managed like trauma patients first, including appropriate resuscitation, as well as activation of trauma teams and obtaining all trauma scans and laboratory studies per institutional protocols. Consider transfer to an appropriate trauma center or center with child abuse expertise if indicated.

If transfer is needed, patients should be sent by emergency medical services or with the police and never in private vehicles.

Notification of police and child protective services is required by law for mandated reporters, including physicians, nurses, social workers, teachers, etc. Reasonable suspicion of abuse meets the legal requirement; abuse does not have to be confirmed. Physicians and other health care providers can be prosecuted for failure to report suspected child abuse.

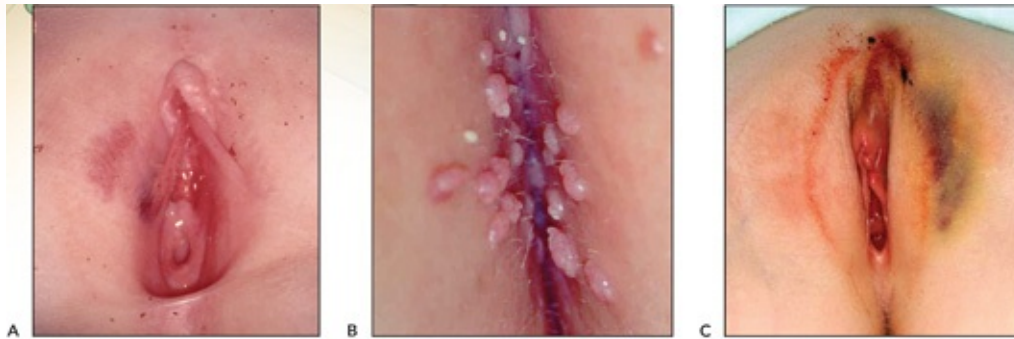
Because abuse is a local police and child protective services investigation, early reporting is important, even if a child is being transferred to another institution.

Child protective services may take a report by phone or may assume custody of the child in the emergency department. If there are any concerns or questions in the emergency department, the child should be admitted.



## 4. CHILD ABUSE—SEXUAL

Stephen John Cico



**FIGURE 1.** A: Bruising in a 2 year old after being with babysitter. B: Anal condyloma in a 6 year old (over age 2 is concerning of abuse). C: Witnessed straddle injury is common and not a sign of abuse. (Courtesy of Becky Wiester, MD.)

### Clinical Presentation

Sexual abuse may involve physical contact with or without penetration (oral, vaginal, anal) or no physical contact (watching pornography or sexual acts, photography, videoing).

The vast majority of pediatric sexual abuse occurs by perpetrators who are known to the child.

Delayed disclosures of episodes of sexual abuse remain common among pediatric patients.

Abnormal sexual behaviors by a child (simulating sexual act, drawing genitals on pictures of people) may indicate abuse but should not be confused with normal behaviors during development (undressing in front of other children, trying to watch others undress, touching a child's genitals).

Children may also present with vague complaints such as abdominal pain, encopresis, genital pain, changes in school performance, or sleep disturbances (although night terrors are normal in early childhood).

### Diagnosis

A normal examination of a child does not exclude sexual abuse, so it is

important to never document information that may be misused in a legal case in the medical record.

Gather information from caregivers and from the child. If the child has disclosed sexual abuse to the parents, then details should be gathered from the parents. If the child makes a disclosure to you, document it, preferably quoting the child.

If the child is developmentally appropriate, open-ended questions should be used. However, it is not incorrect to defer questioning to when the child is evaluated by a physician with experience in abuse.

Examination involves recognizing injuries that need immediate attention, documenting normal and abnormal findings on exam or behaviors witnessed.

## Management

As a mandated reporter, notifying police and child protective services is required if abuse is reasonably suspected; abuse does not have to be confirmed to fall under this obligation.

A forensic examination with evidence collection may be indicated.

Consultation with a child abuse physician can help make this determination but is usually recommended if the alleged abuse occurred within 72 hours but may be indicated even up to 120 hours.

Appropriate prophylaxis for sexually transmitted infections and HIV should be considered. Pregnancy testing and emergency contraception are performed if indicated.

Children will often need comprehensive follow-up, including psychosocial and school-related interventions.

## 5. CRADLE CAP

Lindsay R. Lavin, Cristina Estrada



FIGURE 1. Infantile seborrhea dermatitis, cradle cap.

### Clinical Presentation

“Cradle cap” is infantile seborrhea dermatitis seen on the scalp and sometimes involving the face.

Occurs in infants younger than 1 year, peaks around 3 months of age

Patient is well-appearing and asymptomatic other than skin findings.

### Diagnosis

Clinical diagnosis based on physical exam; no diagnostic testing available

Classic exam findings are greasy scales on the scalp, typically on the frontal region and often involving the forehead and eyebrows.

### Management

Resolves spontaneously within weeks to months; if persists past 1 year of age, needs referral

Recommend conservative treatment for cosmetic purposes.

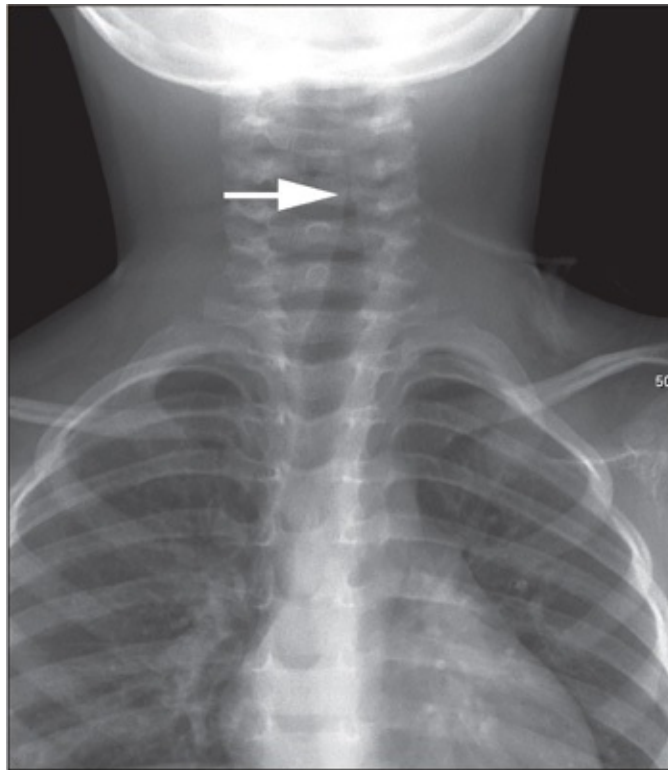
Regularly apply mineral oil to scalp overnight and then gently remove scale with soft brush or comb.

Frequent washing with mild nonmedicated shampoo and gentle removal of scale with soft brush or comb

For more severe cases, may consider selenium sulfide shampoo or topical hydrocortisone cream but typically unnecessary

## 6. CROUP

Stuart A. Bradin



**FIGURE 1.** Steeple sign of croup (arrow).

### Clinical Presentation

Acute respiratory illness which may be associated with a fever and presents with a characteristic “seal like” or barking cough and possibly stridor  
Peak incidence is 6 months to 3 years of age; seasonal, typically fall and winter

Symptoms are typically worse when crying or agitated and at nighttime.  
Self-limited—lasts 5 to 7 days, peaking day 2 to 3 of illness

Most cases mild to moderate; can present as severe distress due to upper airway obstruction

## Diagnosis

Croup is a clinical diagnosis.

The most predominant etiology is parainfluenza type 1, but other viruses may also be causative.

Labs and radiographs are not routinely indicated unless another etiology (e.g., foreign body) is considered. In croup, radiographs may show the steeple sign (subglottic space narrowing).

## Management

*Dexamethasone* 0.6 mg/kg (maximum dose 15 mg) given orally or intramuscularly

If significant distress or stridor at rest, administer racemic epinephrine (2.25% 0.5 mL in 2.5 mL of saline).

Heliox for severe cases to avoid intubation

Humidified air and cool mist only anecdotally beneficial

Admit if persistent stridor/distress, multiple racemic epinephrine treatments required, suspect tracheitis, impending respiratory failure, heliox, or need for mechanical ventilation.



## 7. DIAPER DERMATITIS

Cristina Estrada, Danielle Mercurio



**FIGURE 1.** Diaper dermatitis spares the inguinal folds.

### Clinical Presentation

Most common form of contact dermatitis among children younger than the age of 2 years

The underlying cause of all forms of diaper dermatitis is prolonged contact with moisture.

Both feces and urine contain alkaline substances, which penetrate through the epidermal skin layers, causing inflammation and occasionally skin breakdown.

### Diagnosis

Spares the inguinal folds, as the moist diaper spares direct contact with skin in



these areas

Feces cause erythema and sometimes ulceration of tissue in the perianal area.

Urine commonly affects the thighs, waist band, and genitalia directly.

Other considerations include candida diaper dermatitis, perianal cellulitis (group A *Streptococcus*), atopic dermatitis, and rare conditions, such as Langerhans cell histiocytosis, congenital immunodeficiencies, and neonatal HIV.

## Management

Prevention of further skin breakdown and inflammation by frequent diaper changes, at least eight per day

Cleansing the skin thoroughly with alcohol-free wipes or warm water will remove urine and feces without burning the affected areas.

Application of barrier ointments will protect macerated skin and prevent further contact with urine and feces to promote healing.

Allowing skin to air dry before applying ointments may be helpful.

## 8. FEVER <2 MONTHS OF AGE

Angela C. Maxwell, W. Ricks Hanna, Jr.

Febrile Infant $\leq 2$ Months of Age Recommended Evaluation		
Evaluation	Neonate <30 Days	Young Infant 1–2 Months
CBC with diff	X	X
CMP	X	X
Urinalysis	X	X
Chest x-ray	X (if symptomatic)	X (if symptomatic)
Stool studies	X (if symptomatic)	X (if symptomatic)
Urine culture	X	X
Blood culture	X	X
CSF studies	X	X (if high-risk or initiating antibiotics)
HSV studies	X (if risk factors are present)	

FIGURE 1. Febrile neonate chart evaluation.

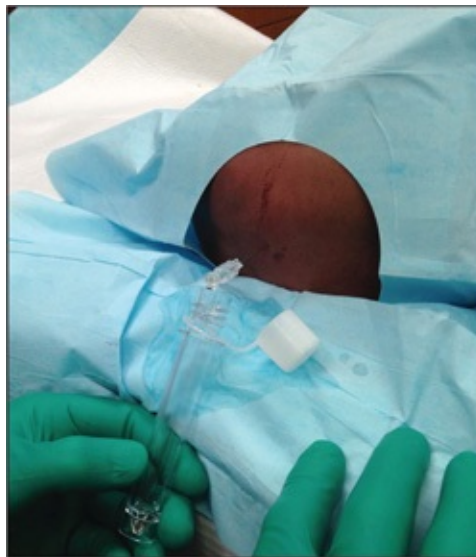


FIGURE 2. Lumbar puncture being performed as part of the standard fever workup of a child younger than 4 weeks of age.

## Clinical Presentation

Rectal temperature  $\geq 38.0^{\circ}\text{C}$  or  $100.4^{\circ}\text{F}$

Pediatric fever comprises an estimated 20% of pediatric emergency department visits.

Physical examination can be unreliable in determining a specific source or determining illness severity.

Rate of serious illness in young febrile infants is between 8% and 12%.

Etiologies include bacteria (e.g., Group B *Streptococcus*, *Escherichia coli*) and viruses (e.g., herpes simplex).

## Diagnosis

0–30 day old: CBC, CMP, UA and urine culture (catheterized), blood culture, cerebrospinal fluid (cell count and differential, glucose/protein, gram stain and culture), respiratory viral testing, CXR and stool culture if respiratory or GI symptoms, respectively

30–60 days old: same work-up and risk stratify to decide on need for lumbar puncture; low risk if previously healthy, full-term, well appearing, no hospitalizations, no underlying or chronic disease, no prior antibiotics, no focal exam findings, UA  $<10$  WBC/HPF, stool  $<5$  WBC and no blood, WBC count 5 to 15,000, absolute band count  $<1500/\text{mm}^3$ , CXR negative (if obtained based on symptoms).

## Management

0–30 days: antibiotics (cefotaxime 100 mg/kg and ampicillin 100 mg/kg) and admission; acyclovir 20 mg/kg if suspect HSV

30–60 days: If low risk, discharge home with 12- to 24-hour follow-up and parent has a reliable phone number and transportation. Do not initiate antibiotics (ceftriaxone 50 mg/kg) unless CSF studies are obtained. Admit for observation if unreliable social situation. If high-risk features, perform lumbar puncture, administer antibiotics (ceftriaxone 100 mg/kg and ampicillin 100 mg/kg), and admit.

## 9. FEVER >3 MONTHS OF AGE

Karen Mangold



FIGURE 1. Infant with fever before antipyretics.



FIGURE 2. Same infant 30 minutes after antipyretics.

### Clinical Presentation

Fever is a very common presenting symptom in pediatrics, accounting for 10% to 20% of pediatric visits to emergency departments. Multiple studies in the postvaccination age show the risk of occult bacteremia without a source is <1%.

### Diagnosis

Consider targeted workup for symptoms, including strep testing and chest x-ray for pneumonia.

Consider a urine culture for females <2 years and males <6 months if child has fever without a source >2 days.

Complete blood count and blood culture are not warranted in immunocompetent children with fever <5 days.

Consider workup for Kawasaki disease if fever persists for 5 days and child has other criteria.

## Management

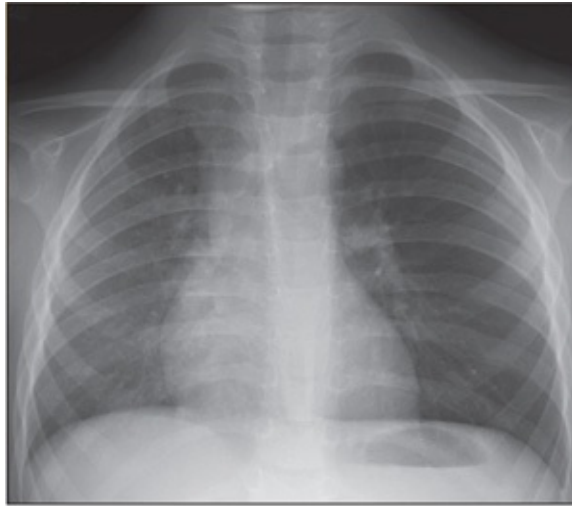
Proper dosage of antipyretics. Instructions to parents should explain that fever is not dangerous, but antipyretics are important for comfort.

Prescribe antibiotics based on most recent recommendations for acute otitis media, pneumonia, and strep. Antibiotics are not warranted for sinusitis in pediatrics.

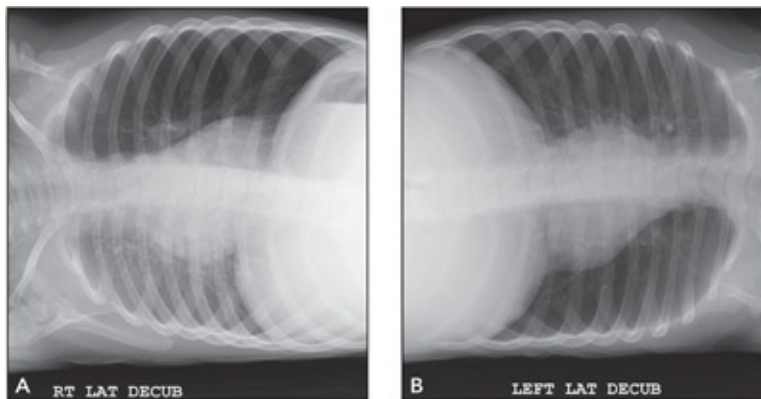
Arrange follow-up with primary physicians if fever continues.

## 10. FOREIGN BODY ASPIRATION

Stephen John Cico, Russ Horowitz



**FIGURE 1.** Hyperinflation of the left lung due to air trapping behind an aspirated pumpkin seed.



**FIGURE 2.** Decubitus films demonstrating normal compression of the lung on the right (A) and lack of left lung compression due to a foreign body (B).

### Clinical Presentation

Foreign body aspiration is most common between 6 months and 4 years of age.

Foods with a round shape (such as peanuts) and balloons are commonly involved in fatal aspirations.

Objects may lodge in the airway without causing significant distress or they may partially or completely occlude the airway.

When below the carina, objects more commonly are found in the right mainstem bronchus.

## Diagnosis

Children may present with coughing, wheezing, shortness of breath, distress, or apnea.

The presentation may be delayed, and misdiagnoses such as “persistent” pneumonia, focal wheezing, and “new-onset” asthma may be made initially. If the child is stable, radiographs may be obtained to localize the object, although organic material will not be visible. Clues of aspiration may include atelectasis, hyperinflation, mediastinal shift, lobar collapse, or postobstructive infiltrates.

No findings on radiographs should never be used to exclude aspiration of organic or non-radiopaque objects.

## Management

Back blows alternating with chest compressions in infants or abdominal thrusts (the Heimlich maneuver) are the initial treatment.

For objects lodged above the glottis, direct visualization and removal with McGill forceps may be successful, but blind sweeps of the mouth are not recommended.

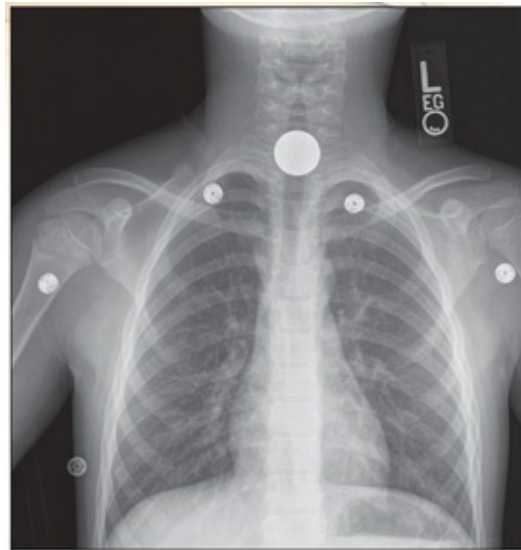
Children should be kept in a position of comfort, often in the parent’s lap, with minimal agitation if presenting with a partial obstruction. Radiographs and intravenous access should not delay transfer to either a facility with bronchoscopy or to the operating suite.

Children who are well-appearing but had a witnessed choking episode, even with normal radiographs, should be seen in follow-up by an otolaryngologist because bronchoscopy may still be usually warranted.



## 11. FOREIGN BODY INGESTION

Stephen John Cico, Russ Horowitz



**FIGURE 1.** Coin in the upper third of the esophagus, which is unlikely to pass.



**FIGURE 2.** Coin in the stomach.

### Clinical Presentation

Foreign body ingestions are most common between 6 months and 4 years of

age.

Coins are the most commonly ingested foreign body, followed by fish bones. History may include choking without respiratory distress; some children may complain of soreness or pain in the throat or chest. Drooling, inability to swallow, or respiratory distress suggests ingestions where the foreign object is lodged in the esophagus or aspirated into the airway.

## Diagnosis

Radiographs that include the neck, chest, and abdomen localize radiopaque foreign bodies.

Coins in the esophagus appear round on the AP radiograph, while those in the trachea are perpendicular.

Ultrasound and computed tomography are useful for localizing non-radiopaque objects (e.g., plastic).

## Management

Foreign bodies lodged in the upper third of the esophagus often cause more respiratory symptoms and require endoscopic removal. Foreign bodies in the mid to distal esophagus may spontaneously pass; these children often are admitted overnight and reevaluated in the morning. Foreign objects that are identified in the stomach or intestines will usually spontaneously pass.

Magnets are a specific concern because they can attract to each other across the mucosa of the intestines and cause necrosis and perforation. Removal of more than one magnet is recommended.

Sharp, elongated objects pose a risk of perforation, particularly when >4 to 6 cm (e.g., sewing needles). Consider endoscopic removal.

Button batteries can be mistaken for coins on radiographs, although the double-rim contour is often identified. Alkali-induced liquefaction necrosis or corrosion can occur when lodged in the esophagus and necessitate urgent endoscopic removal. Button batteries that have passed into the stomach can be observed for passage, with intervention if complications arise.

If children experience vomiting, abdominal pain, or distention, they should be instructed to return; fistulas, obstruction (sometimes at the ileocecal valve),

and perforation are rare complications.

## 12. FUSSY BABY—HAIR TOURNIQUET

Jocelyn A. Plesa, Robert W. Hickey



FIGURE 1. Hair tourniquet, closeup.

### Clinical Presentation

Unexplained crying and fussiness in infants can be the result of a corneal abrasion, incarcerated hernia, or hair-thread tourniquet constricting the finger, toe, or penis.

### Diagnosis

Remove all constricting clothing, jumpers, and mittens to evaluate digits. Edematous tissue encircling the digit with a fine line of demarcation represents a hair-thread tourniquet.

Other considerations include nonaccidental trauma, toddler's fracture in ambulatory children, otitis media, teething, and sepsis.

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## Management

Apply depilatory cream for 10 minutes if the constricting agent is hair. If above is unsuccessful or if the material is not hair, use a blunt probe to isolate the fiber from the underlying tissue and unwind the fibers or cut them with fine-tipped scissors.

If above techniques are unsuccessful, perform digital nerve block; prepare digit in sterile fashion; incise affected digit in a perpendicular fashion to the constricting thread at the 3 o'clock, 9 o'clock, or 12 o'clock position.

Evaluate neurovascular status post removal.

## **13. GREENSTICK FRACTURES**

Cristina Estrada, Danielle Mercurio



**FIGURE 1.** AP greenstick fractures.



**FIGURE 2.** Lateral greenstick fractures.

## Clinical Presentation

Most common fracture pattern seen in children, accounting for 50% of fractures in children younger than 12 years due to softer, more pliable bones  
Highest incidence between 7 and 12 years  
Commonly involves the radius, ulna, or clavicle

## Diagnosis

Incomplete fractures involving the diaphyseal–metaphyseal junction of long bones  
Intact cortex on one side with disrupted cortex and periosteum on the other side  
Most commonly diagnosed on anteroposterior (AP) and lateral views of affected limb, with notable angulation and rotation  
May be difficult to diagnose because there may not be much pain or swelling, disuse, or reduced range of motion



Mild greenstick fractures sometimes are thought to be sprains.

## Management

Pain management and initial stabilization for comfort is essential while films are obtained.

Commonly requires closed reduction by either a well-trained emergency physician or orthopedic consultant

Two methods of reduction are used. The rotational method is easier to perform and less painful for the patient. However, the fracturing to completion method has a lower refracture rate.

Immobilization with splinting or casting is essential for proper healing, with follow-up scheduled in 1 week with an orthopedist and parents should be given instructions for close monitoring for compartment syndrome.

## 14. HAND-FOOT-MOUTH DISEASE

Rebecca Kidd, Cristina Estrada



**FIGURE 1.** Small vesicles on an erythematous base on the palm.



**FIGURE 2.** Similar lesions seen on the plantar aspect of the foot.

## Clinical Presentation

It usually occurs in epidemics in summer to early fall and is caused by coxsackie or enteroviruses.

Disease begins with a prodrome of low-grade fever, sore mouth, malaise, and anorexia, which is followed 1 to 2 days later with oral lesions, followed by skin lesions.

Oral lesions occur in the anterior portion of the mouth (buccal mucosa, gingivae, tongue, soft palate, uvula, and anterior tonsillar pillars) and are characterized by small, shallow ulcers with surrounding erythematous halo.

Cutaneous lesions start as erythematous macules that progress to small vesicles on an erythematous base. They are usually found on palmar aspects of hands and fingers and plantar surfaces of feet and toes. Lesions can be mildly tender or pruritic.

## Diagnosis

This is a clinical diagnosis, and there is rarely need for laboratory testing. Skin involvement is not necessary to make the diagnosis. Ninety percent of

cases will have oral involvement; two-thirds will have cutaneous lesions. Usually self-limited with lesions lasting 2 to 7 days; the vesicles will heal without scarring.

## Management

Management is supportive and symptomatic care, including antipyretics and analgesics.

Evidence is mixed on use of topical analgesics, such as viscous lidocaine or combination of Maalox and diphenhydramine.

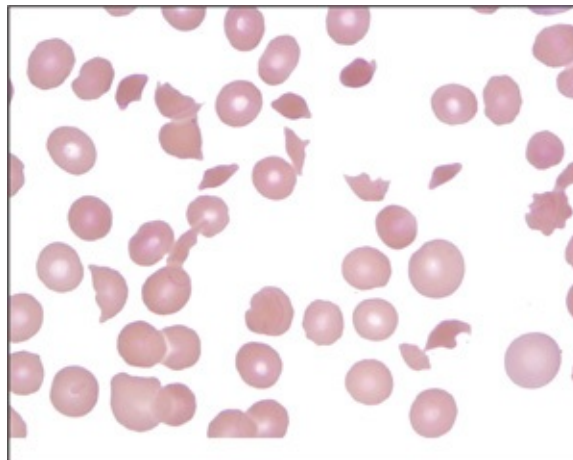
Adequate hydration is the most important factor. Caregivers should offer a soft diet and cool liquids, avoiding acidic and citrusy beverages.

## 15. HEMOLYTIC UREMIC SYNDROME

Angela Zamarripa, Michele Carney



**FIGURE 1.** Petechiae and pupura in a patient with HUS.



**FIGURE 2.** A remarkable number of schistocytes are present in this patient with microangiopathic hemolytic anemia due to hemolytic uremic syndrome. (From Pereira I, George TI, Arber DA. *Atlas of Peripheral Blood*. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.)

### Clinical Presentation

Hemolytic uremic syndrome (HUS) peaks in June through September. Multiple organisms are implicated including *Shigella dysenteriae* type 1, *Salmonella*, *Yersinia*, and most commonly Shiga toxin–producing

enterohemorrhagic *Escherichia coli*, specifically *E. coli* O157:H7. Outbreaks do occur, but only 10% of cases arise from epidemics. Transmission routes include swimming pools, person-to-person contact (i.e., daycare), animal contact, foodborne, or drinking water–related. Forty percent to 60% of patients will require dialysis; however, the mortality rate has drastically decreased since the 1950s.

## Diagnosis

Characterized by a triad of microangiopathic hemolytic anemia, thrombocytopenia, and oliguric renal failure. Symptoms can include hypovolemia, pallor, listlessness, lower extremity bruising, abdominal pain, vomiting, and colitis, which is often hemorrhagic. Laboratory studies will demonstrate leukocytosis, reticulocytosis, transaminitis, and elevated lactate dehydrogenase and bilirubin. Coagulation studies will be normal, and a blood smear will show red blood cell (RBC) fragments and schistocytes. Urinalysis will be positive for heme, protein, bilirubin, and white blood cells. Stool culture should be sent.

## Management

Supportive care is key, starting with isotonic fluid resuscitation. If oliguric, a trial of furosemide can be administered after intravascular volume is restored. Antibiotics and antimotility agents should be avoided. Sustained hypertension should be treated with vasodilators such as hydralazine or calcium channel blockers. Packed RBC transfusions for symptomatic anemia or hematocrit <18% to 20%. Platelets are indicated only if there is active bleeding.



## 16. HENOCCH-SCHÖNLEIN PURPURA

Kelly Black, Alexandra Schaller



FIGURE 1. Henoch-Schönlein purpura in an adolescent.

### Clinical Presentation

Immunoglobulin A–mediated small vessel vasculitis that affects the skin, intestines, kidneys, and joints; exact etiology is unknown.

Most common in prepubescent children; incidence higher in males

Maculopapular purpura, most commonly on lower extremities and buttocks

Fever, headache, arthralgias, arthritis, spasmodic abdominal pain, diarrhea, hematuria, glomerulonephritis, Hemoccult positive stool, intussusception

### Diagnosis

Diagnosis is based on typical skin, joint, and kidney findings; if characteristic findings are not present, this is a diagnosis of exclusion.

Laboratory testing not required for diagnosis; urinalysis, inflammatory markers, and kidney function may suggest the diagnosis.



If concern for intussusception, abdominal radiography and/or ultrasound should be obtained.

## Management

Symptomatic treatment including fluids and pain control with nonsteroidal antiinflammatory drugs

If gastrointestinal symptoms or kidney involvement are present, corticosteroids may be included in treatment.

Prognosis is good if no renal involvement as Henoch-Schönlein purpura is usually a mild illness that resolves spontaneously; if renal involvement is suspected, then a biopsy may be needed for diagnosis and prognosis.

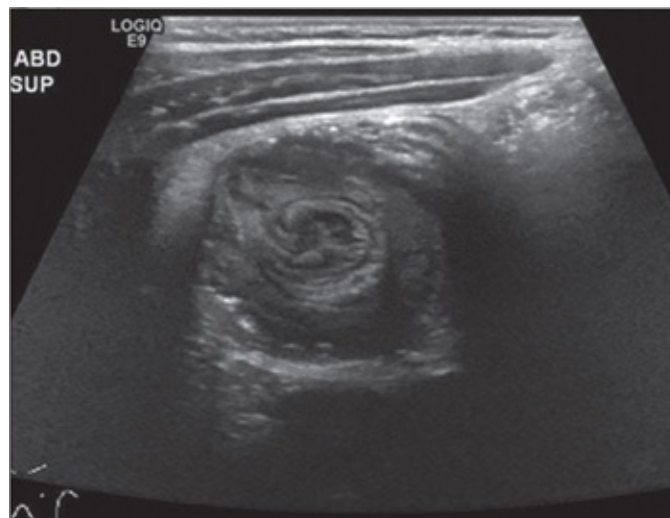
Usually resolves after 4 to 6 weeks; relapse can occur.

## 17. INTUSSUSCEPTION

Ellen G. Szydlowski, Jennifer R. Marin



**FIGURE 1.** Paucity of air in the right lower quadrant (arrow) and the crescent sign (asterisk).



**FIGURE 2.** Target sign of intussusception on ultrasound.

### Clinical Presentation

Intussusception is the most common cause of intestinal obstruction in children between 3 months and 6 years of age (peak incidence between 5 and 9

months).

Children usually present with bilious emesis and colicky abdominal pain.

Infants may present with somnolence alone.

Classic triad of abdominal pain, currant jelly stool, and a palpable right upper quadrant mass may only be present in up to 40% of patients.

## Diagnosis

Rectal exam positive for occult blood in 50% of patients with intussusception

Posteroanterior and upright or left lateral decubitus abdominal x-rays may show paucity of air in the right lower quadrant, target sign, or crescent sign.

Abdominal ultrasound is the imaging modality of choice and demonstrates the “target sign” in transverse orientation and the “pseudokidney sign” in longitudinal orientation.

## Management

If ultrasound is unavailable or if ultrasound identifies an intussusception, the patient should undergo air or contrast enema, which is both diagnostic and therapeutic.

If reduction is unsuccessful or there are signs of perforation, manual reduction is required in the operating room.

Risk of recurrence is up to 15% in patients undergoing nonoperative reduction and up to 3% after operative reduction, usually within the first 24 hours.

Admission and 24-hour observation after successful reduction is recommended.

## 18. KAWASAKI DISEASE

Jason Lowe



FIGURE 1. A: Limbic sparing conjunctivitis. B: Strawberry tongue and cracked lips. C: Red foot. D: Polymorphic rash. E: Coronary aneurysms.

### Clinical Presentation

Kawasaki disease, also known as *mucocutaneous lymph node syndrome*, is a systemic vasculitides of uncertain etiology. Overall, the acute symptoms are self-limited. However, if left untreated, 20% will develop coronary artery aneurysm. With treatment (within 2 weeks of start of fever), that risk drops to 4%.

Incidence of 20 per 100,000 in the United States with the majority less than 5 years old

### Diagnosis

Diagnosis is made clinically and *requires* fever for at least 5 days with no other source.

Additionally, four of the following five criteria must be present: bilateral limbic sparing conjunctivitis, oral mucosal changes (injected/cracked lips, strawberry tongue), extremity changes (red hands/feet, desquamation of digits), polymorphic rash, cervical lymphadenopathy

In unclear cases, supporting laboratory results may help elucidate the diagnosis but are nonspecific. These include an elevated erythrocyte sedimentation rate and C-reactive protein, leukocytosis with left shift, thrombocytosis, anemia, sterile urethral pyuria (clean catch or bag specimen), elevated liver function tests, and hyponatremia.

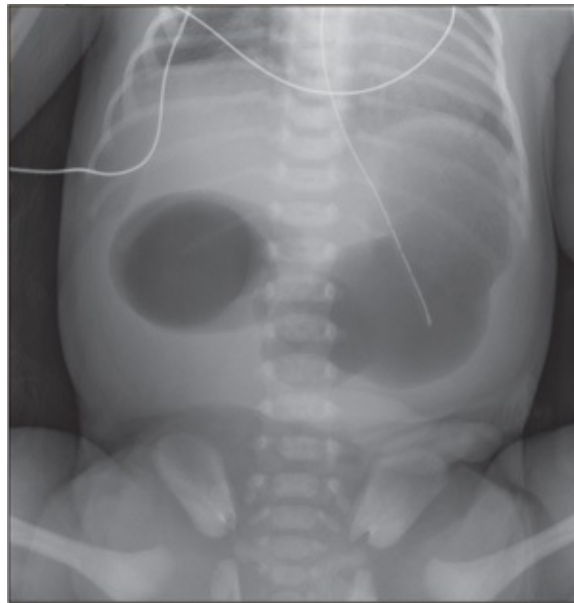
## Management

Consultation with appropriate subspecialist for treatment and monitoring is recommended.

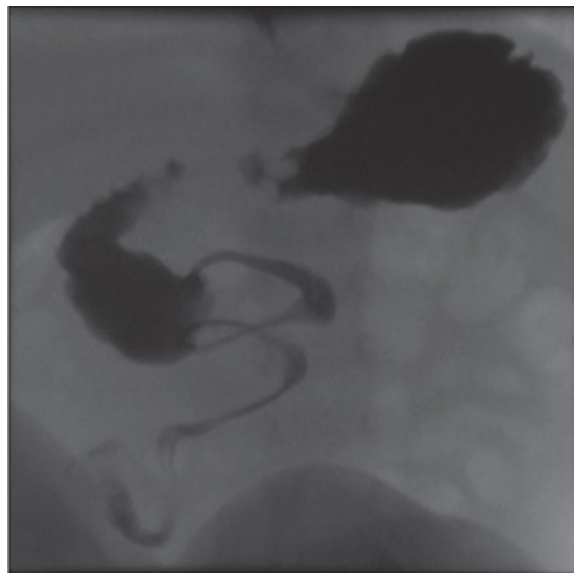
Treat with intravenous immunoglobulin 2 mg/kg for one dose and aspirin 100 mg/kg/day in four divided doses.

## 19. MALROTATION

Lauren M. Mutter, Rudy J. Kink



**FIGURE 1.** Malrotation on plain radiograph. Note the "double bubble" sign.



**FIGURE 2.** Malrotation on upper GI demonstrating corkscrew appearance of the small intestine suggesting midgut volvulus.

Clinical Presentation

Intestinal malrotation is present in 1 per 6,000 births; incidence of males to females is 2:1.

A disease of infants, it is rare in children older than 1 year.

Caused by rotational abnormality of intestines during embryonal development

Should be considered in any infant who presents with bilious or nonbilious emesis, abdominal distention, or bloody stool

Malrotation may be complicated by volvulus, which is life-threatening ischemia of the bowel.

## Diagnosis

Diagnosis is made using plain films followed by an upper gastrointestinal (GI) contrast study to best visualize the duodenum.

Barium enema and ultrasound can be helpful additions for diagnosis, but if negative, cannot exclude malrotation.

If malrotation is complicated by volvulus and the patient is unstable, emergent surgical evaluation is necessary.

Plain films will show a “double bubble” sign indicative of duodenal obstruction, whereas the upper GI will show a misplaced duodenum with corkscrew appearance.

## Management

Immediate pediatric surgery consultation

Keep NPO, place nasogastric tube to suction, correct electrolytes, and implement fluid resuscitation.

Broad-spectrum antibiotics if possible prior to surgery

Surgical intervention does not correct the rotational abnormality but widens the mesentery and places the bowel in a nonrotation configuration to reduce a potential future volvulus.



## 20. MANAGEMENT OF FORMER PRETERM INFANT

Haifa A. Samra, R. Lane Coffee, Jr., Stephen John Cico



**FIGURE 1.** NG feedings are an option for preterm infants with poor oral intake.

### Clinical Presentation

Bronchiolitis and upper respiratory tract infection: may present with respiratory distress, wheezing, tachypnea, difficulty feeding, cyanosis, or apnea

Necrotizing enterocolitis (NEC): may present with vomiting, abdominal distention, tachycardia, hypotension, pale, prolonged capillary refill time

Fever: often present with fussiness, lethargy, decreased oral intake, feeling warm or sweating, or may present only with an elevated measured temperature

Hyperbilirubinemia: presents with change in skin (jaundice) or eye color (icterus)

Gastroesophageal reflux (GERD): presents with nonbloody and nonbilious vomiting after feedings, possibly poor weight gain

### Diagnosis

Chest radiographs may show a bilateral increase in perihilar markings; sending rapid influenza and respiratory syncytial virus (RSV) tests may be

beneficial to guide management in those <3 months.

Obtain abdominal radiographs to evaluate for evidence of pneumatosis (NEC) in former preterm infants with abdominal distention or pain.

Laboratory studies should be obtained for evaluation of specific diagnoses, such as bilirubin for jaundice.

## Management

Bolus for dehydration with normal saline 20 mL/kg

Consider checking point of care glucose in infants with poor oral intake, vomiting, or lethargy because glucose stores are limited, particularly in preterm infants. Consider nasogastric (NG) feedings as an option for nutritional support of these children.

Some former preterm infants with upper respiratory infections or bronchiolitis may respond to high-flow nasal cannula oxygen.

Relieving upper airway obstruction by nasopharyngeal suctioning in obligate nose breathers may improve respiratory status.

Use of humidified air may be beneficial.

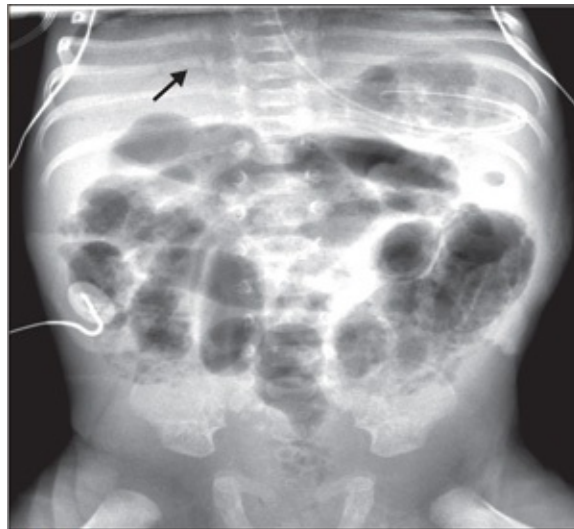
In general, a respiratory rate of 60 breaths per minute or greater or oxygen saturations below 90% on room air are indications for intravenous hydration and admission.

Give small, frequent feedings to infants who may have GERD to keep stomach from over filling and hold infant upright for 30 minutes after feeding.

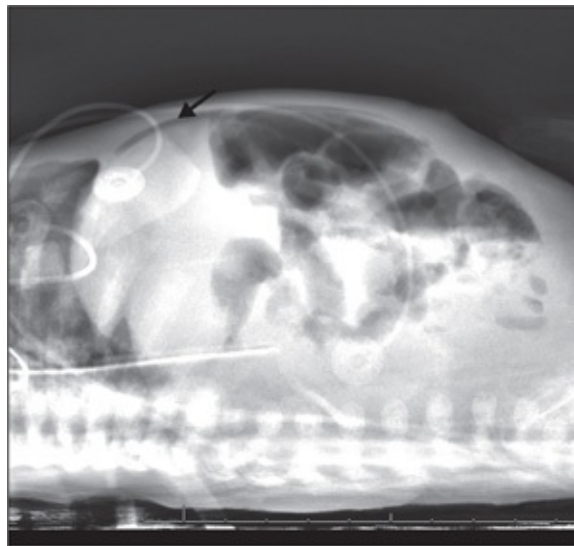
During sleep, raise the head of the infant's crib mattress 6 inches. Acid suppressants such as H<sub>2</sub>-blockers and proton pump inhibitors may be considered in the most severe cases.

## 21. NECROTIZING ENTEROCOLITIS

Stephanie K. Kukora, Andrew N. Hashikawa



**FIGURE 1.** Plain film with portal venous air (arrow) in a patient with NEC.



**FIGURE 2.** Lateral plain radiograph demonstrating free air (arrow) in a patient with perforated NEC.

### Clinical Presentation

Infants present with feeding intolerance, abdominal distention, and bloody stools.

This condition is rare in term newborns, although they comprise approximately 10% of necrotizing enterocolitis (NEC) cases.

NEC should be suspected in symptomatic infants:

Born preterm

Intrauterine growth restricted or small for gestational age

Conditions that impair blood flow such as congenital heart lesions, polycythemia, or clotting disorders

## Diagnosis

Exam is notable for abdominal distention and pain with palpation, although findings are often nonspecific.

Anteroposterior and lateral abdominal radiographs with pneumatosis, portal venous gas, and free abdominal air are diagnostic.

Elevated inflammatory markers (white blood cell and C-reactive protein) further support evidence of infection, and a blood culture may reveal causative organisms and sensitivities.

## Management

Infant should take nothing by mouth, with nasogastric catheter placed to suction.

Obtain vascular access to provide intravenous antibiotics, fluid resuscitation, nutrition, and pressor agents if needed.

Infants require admission to neonatal intensive care unit.

Surgical consultation is warranted; surgical management is necessary if free air is evident on radiographs.

## 22. NEONATAL ACNE AND NEONATAL RASHES

Margaret Wolff, Melissa Skaugset



FIGURE 1. Neonatal acne.

### Clinical Presentation

Onset  $\leq 6$  weeks of age, usually between 2 and 4 weeks of age. Lesions are not present at birth.

Occurs in up to 20% of newborns

Small red bumps or pustules on the face (cheeks, nose, chin, forehead) and less commonly on the back, upper chest, or neck

### Diagnosis

No testing is indicated if lesions are characteristic in appearance and age of onset.

Differential of vesiculopustular lesions in neonates includes erythema toxicum neonatorum, transient neonatal pustular melanosis, infantile acropustulosis, infantile acne, neonatal herpes simplex virus, and staphylococcal pyoderma.

## Management

Parental reassurance that it is benign and self-limited; typically resolves within 4 months without scarring

Clean daily with gentle soap and water and avoid exogenous oils and lotions.

Acne medications should not be used.

Follow-up is with a primary care physician.



## 23. OMPHALITIS

Valerie E. Parr, Mark Meredith



**FIGURE 1.** Omphalitis with tissue necrosis and surrounding cellulitis.

### Clinical Presentation

Infection of the umbilicus or umbilical cord stump with erythema and purulent discharge

May be associated with tenderness, induration, or even bleeding

Risk factors include low birth weight, prolonged labor, prolonged rupture of membranes, chorioamnionitis, delivery in developing countries, umbilical catheterization, home birth, or deficiencies of the immune system (such as disorders of leukocyte adhesion, neutrophils, natural killer cells, or interferons).

Symptoms such as fever, poor feeding, irritability, or lethargy suggest a complication. The most common complication is sepsis, which is likely from bacteria spreading via the umbilical vessels.

Most common pathogens include *Staphylococcus aureus*, *Streptococcus pyogenes*, and gram-negative bacteria (*Escherichia coli*, *Klebsiella*, *Proteus*).



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## Diagnosis

Culture of discharge recommended

Complete blood count, blood culture, catheterized urinalysis and culture, and cerebrospinal fluid studies and cultures should be performed if there are signs of sepsis.

## Management

Antibiotics to cover both gram-positive (plus methicillin-resistant *Staphylococcus aureus* coverage in high prevalence areas) and gram-negative bacteria

Anaerobic coverage with clindamycin or metronidazole is needed if particularly foul-smelling discharge or a history of chorioamnionitis.

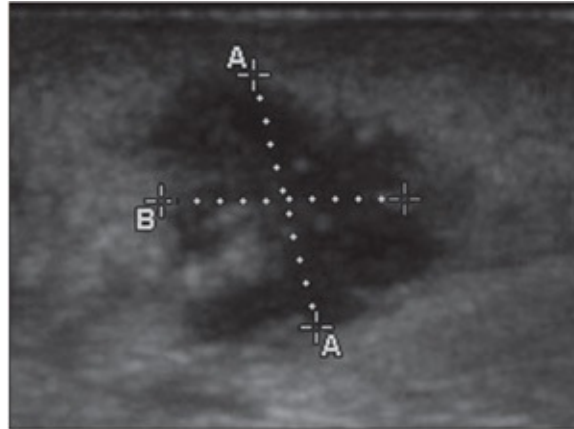
Neonates/infants will require intravenous antibiotics and admission. Older patients without any systemic signs can possibly be treated with oral antibiotics.

## 24. PEDIATRIC ABSCESS

Derya Caglar



**FIGURE 1.** Image of soft tissue abscess on arm. (From Burkhardt C, Morrell D, Goldsmith LA, et al. *VisualDx: Essential Pediatric Dermatology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.)



**FIGURE 2.** Ultrasound image of skin abscess.

### Clinical Presentation

Cellulitis and abscesses are a common complaint in the pediatric population, with a sharp increase during the last 15 years matching the emergence of methicillin-resistant *Staphylococcus aureus* (MRSA).

Prevalence of MRSA in soft tissue infections varies significantly with geography, ranging from 15% to 80% of all soft tissue infections.

Risk factors include skin trauma, overcrowding, history of frequent antimicrobial use, and exposure to health care providers or known MRSA contacts.

The exam is often significant for erythema, edema, induration, significant tenderness to palpation, and occasionally purulent drainage, although exam alone cannot delineate between MRSA and methicillin-sensitive *S. aureus*.

## Diagnosis

Clinical examination and history are often sufficient to make the diagnosis, although wound culture is necessary to confirm MRSA status.

Blood work is not recommended in most cases, unless there is concern for systemic illness or deeper infection (i.e., necrotizing fasciitis).

An ultrasound can be helpful in determining the presence of an abscess and guiding need for incision and drainage (I&D).

## Management

Simple abscesses with minimal surrounding cellulitis can be treated with thorough I&D alone; no antibiotics are needed in otherwise healthy patients even when due to MRSA.

When the abscess is associated with significant cellulitis or systemic illness, antibiotics with adequate empiric MRSA coverage (i.e., clindamycin) should be initiated.

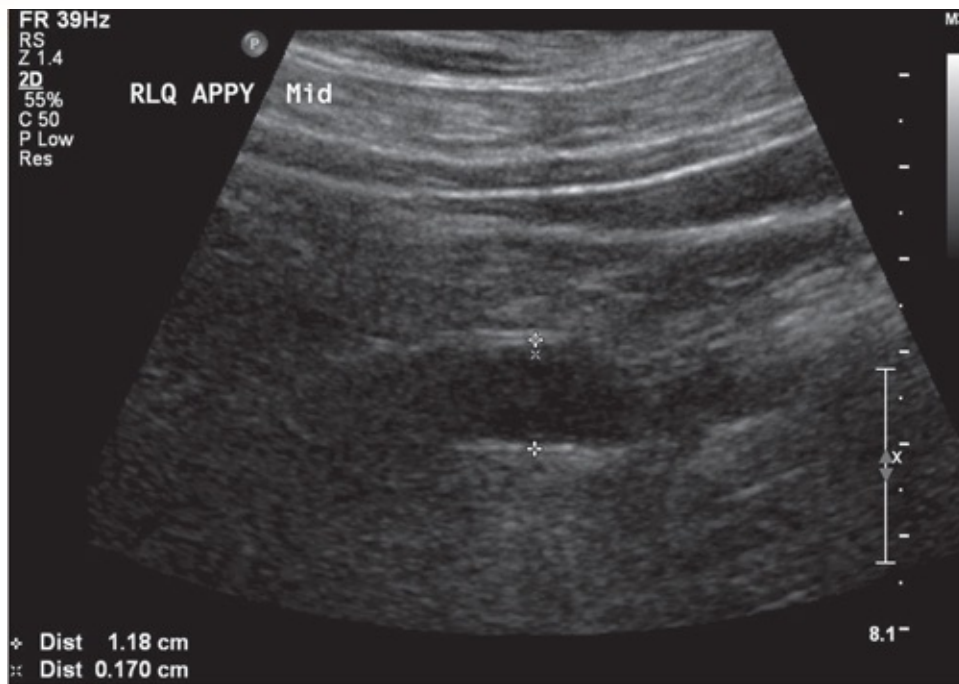
In areas with high clindamycin resistance or known patient history of resistance, trimethoprim-sulfamethoxazole is an option for outpatient treatment.

Patients should have close primary care provider follow-up within 2 to 3 days to assess for improvement, review wound culture results, or need for further treatment.

Admission and intravenous antibiotics should be considered for extensive disease, signs of systemic illness, failed outpatient treatment, or any concern for necrotizing fasciitis or complication.

## 25. PEDIATRIC ACUTE APPENDICITIS

Derya Caglar



**FIGURE 1.** Ultrasound image of a noncompressible enlarged appendix in a patient with appendicitis.

### Clinical Presentation

Appendicitis is the most common condition in children requiring emergency abdominal surgery and is diagnosed in approximately 8% of children presenting with abdominal pain.

It presents most frequently in the second decade of life and is more common in boys than girls.

Classic examination findings include anorexia, initial periumbilical abdominal pain that migrates to the right lower quadrant within 24 hours of onset, vomiting, and fever.

Young patients often present with perforation due to less classic, nonspecific symptoms, difficulty eliciting an accurate history, and a difficult assessment and examination.

## Diagnosis

Clinical examination and history can often make the diagnosis with imaging as an aid in equivocal cases.

Elevated complete blood count and C-reactive protein are often seen in appendicitis but are nonspecific findings.

Positive findings on ultrasound include a noncompressible and enlarged tubular structure in the right lower quadrant, wall thickening, presence of a fecalith, and free fluid.

Computed tomography with contrast and magnetic resonance imaging can also be used to confirm diagnosis when ultrasound is not available or unable to visualize the appendix.

## Management

Definitive care involves surgical removal of the appendix.

Intravenous hydration, pain control, and antibiotics should be administered preoperatively while awaiting surgery.

Complications, including intraabdominal abscesses, small bowel obstruction, and prolonged hospital stay, are much more common in cases with perforation.

## 26. PEDIATRIC ASTHMA

Margaret Wolff

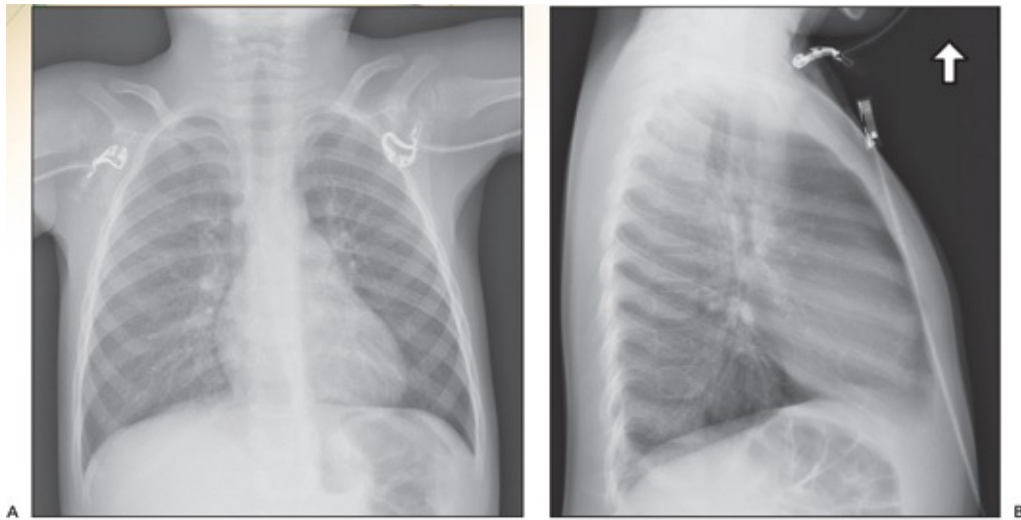


FIGURE 1. A: AP. B: Lateral chest radiographs demonstrating hyperinflation and peribronchial cuffing.

### Clinical Presentation

Approximately 10% of children in the United States have asthma. Acute asthma exacerbations occur due to a reduction in airway luminal diameter due to airway inflammation and reversible bronchoconstriction in response to a specific trigger. Upper respiratory infections are the most common trigger in children.

Exacerbations consist of cough, wheezing, dyspnea, and chest tightness. Patients may have hypoxia or hypercapnia.

### Diagnosis

Acute airway obstruction that is reversed following bronchodilator therapy is suggestive of the diagnosis of asthma.

Chest radiographs are not necessary for the majority of children with wheezing. If performed, radiographs may demonstrate hyperinflation of lungs with peribronchial cuffing.

Chest radiographs should be considered in patients with high fever, focal exam findings, or severe disease or if they are not responding to therapy as expected.

## Management

Reverse airflow obstruction by administration of repeated doses of inhaled short-acting bronchodilators and early systemic glucocorticoids

Early use of ipratropium bromide 500 µg may help pediatric patients presenting with status asthmaticus.

For severe exacerbations, consider use of intravenous magnesium sulfate, intramuscular epinephrine or terbutaline, and continuous albuterol.

Administer supplemental oxygen for hypoxemia.

Consider hospitalization for patients who require β-agonists more than every 4 hours, patients with hypoxia, or patients with inadequate access to medical care for follow-up or worsening exacerbation.



## 27. PEDIATRIC CONSTIPATION

Jason Lowe



**FIGURE 1.** Plain radiograph demonstrating constipation.

### Clinical Presentation

Constipation is a common cause of abdominal pain in pediatric patients, representing 3% to 5% of pediatric emergency visits. The vast majority are due to functional causes, and only 5% are due to organic.

May be acute or chronic; often, patient is described as being in severe pain at home.

History may not always indicate any issue with stooling or may be positive for loose or watery stool.

May mimic more severe etiologies of abdominal pain; concerning symptoms include poor weight gain, stooling issues since birth, vomiting, fever, associated urinary incontinence, and other extraintestinal symptoms. More concerning physical signs include severe abdominal tenderness or distension, poor anal tone, absent cremasteric reflex, and other abnormal neurologic findings.

## Diagnosis

High clinical suspicion should be maintained in all children with abdominal pain, although it is a diagnosis of exclusion.

Abdominal masses may not always be palpated.

Usually diffuse pain with no peritoneal signs

Rectal exam may be useful to assess for firm stool in vault.

Abdominal plain films can assess for severity of impaction and rule out obstructive processes, especially when history is unclear.

## Management

For severe impaction, an enema may offer quick relief in the emergency department. Manual disimpaction should be avoided except in the most extreme situations.

Polyethylene glycol powder (MiraLAX) daily for 1 to 2 weeks is useful to eliminate residual stool and decompress bowel. A common “cleanout” regimen includes eight capfuls of polyethylene powder in 64 oz of sports drink consumed over 24 to 48 hours.

Dietary changes, including increased fiber and water intake, along with behavioral modifications, are the keys to prevention of recurrence.

## 28. PEDIATRIC EAR PAIN

Suzanne Schmidt

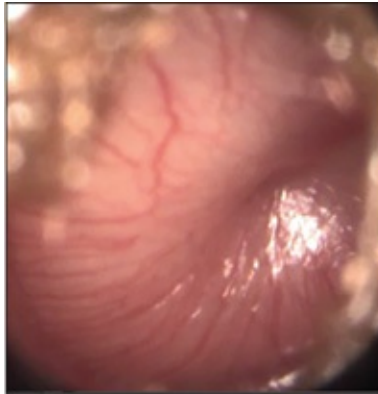


FIGURE 1. Acute otitis media.



FIGURE 2. Otitis media with effusion. (Assets provided by Anatomical Chart Company.)

### Clinical Presentation

Ear pain is one of the most common presenting pediatric concerns.

Not all children with ear pain have an ear infection.

Ear pain can be caused by infected fluid in the middle ear, uninfected fluid in the middle ear, infection of the external auditory canal, impacted cerumen or foreign objects in the ear canal, or may be referred pain from a sore throat, dental infection, or local lymphadenopathy.

Children with acute otitis media (AOM) often present with fever and ear pain

following an upper respiratory tract infection.

If the tympanic membrane perforates, a child with AOM may have purulent ear drainage.

## Diagnosis

AOM is infected fluid in the middle ear and is diagnosed based on otoscopic findings of a bulging tympanic membrane with purulent fluid behind it.

Serous otitis or otitis media with effusion (OME) is when clear or amber fluid is present behind the tympanic membrane. The tympanic membrane may be retracted. This is not an ear infection and does not need antibiotics.

OME (otitis media with effusion) is uninfected fluid in the middle ear that is common in children with an upper respiratory tract infection or after AOM has resolved.

## Management

Treat with analgesics.

AOM can be treated with immediate antibiotic therapy or a “wait-and-see” approach. The wait-and-see approach is when the patient/family is given an antibiotic prescription and told not to start the medication unless symptoms do not improve or worsen within 48 to 72 hours.

Wait-and-see approach is best for children 6 months and older with unilateral AOM, mild ear pain, symptoms <48 hours, and T<sub>max</sub> <39°C. Children older than 2 years with bilateral AOM also qualify for wait-and-see.

Amoxicillin 80 to 90 mg/kg/day divided twice daily for 5 to 10 days (5 to 7 days if older than 2 years).

Augmentin should be prescribed if the patient has received amoxicillin in the last 30 days, has concurrent purulent conjunctivitis (suggesting *Haemophilus influenzae*), or has recurrent ear infections resistant to amoxicillin.

Cefdinir should be prescribed to penicillin-allergic patients without severe life-threatening allergic reactions.

## 29. PERIANAL STREP

Mindy Longjohn, Peter Jones

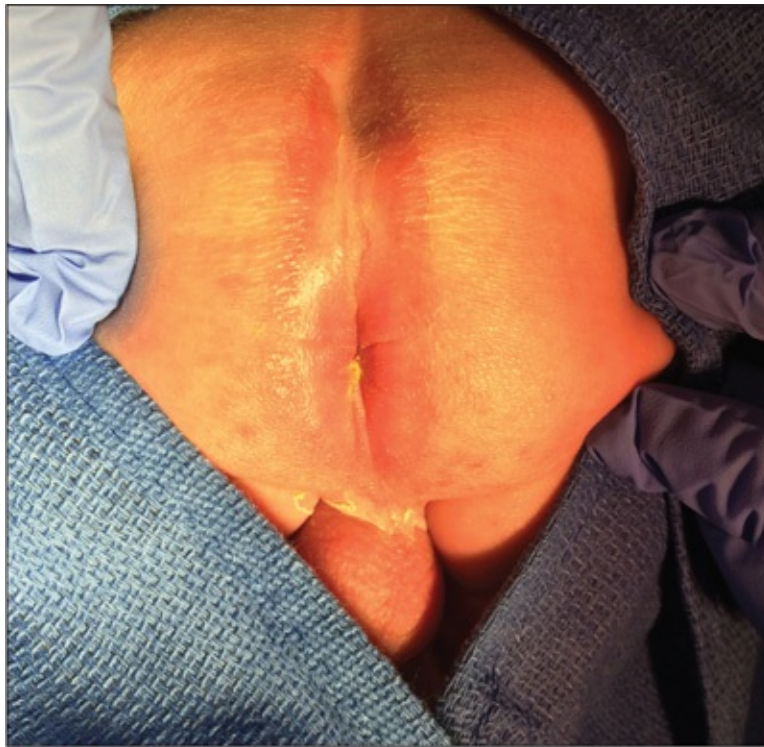


FIGURE 1. Perianal strep.

### Clinical Presentation

Predominantly a disease of early childhood, ranging from infancy to preteen years, but can be seen in adults

Incidence ranges from 1 for every 200 to 2,000.

Most commonly caused by group A  $\beta$ -hemolytic *Streptococcus*

### Diagnosis

Well-demarcated perianal erythematous rash is the most common feature, classically described as “beefy” red but can be mild and pink.

Associated symptoms are pruritus and pain.

Constipation, anal fissures, and blood-streaked stools may be present. Diagnosis is confirmed by the growth from a culture for group A *Streptococcus* from a swab of the erythematous area; although not approved for diagnosis, rapid strep testing may be positive.

## Management

Antibiotic recommendation: oral penicillin (50,000 to 100,000 U/kg) Amoxicillin (40 mg/kg divided twice a day) for 10 days is often used. Erythromycin (20 to 40 mg/kg/day in two to four divided doses) or clindamycin (30 to 40 mg/kg in three divided doses) may be used for penicillin-allergic patients. Alternative may be cefuroxime (20 mg/kg divided twice a day) for 7 days. Recurrence has been shown to be up to 36% to 43%.



## 30. POSTSTREPTOCOCCAL GLOMERULONEPHRITIS

Mark Snider, Mark Meredith



FIGURE 1. Gross hematuria in glomerulonephritis.



FIGURE 2. Red cell casts under microscopy (arrow).

### Clinical Presentation

The leading cause of glomerulonephritis in children, rarely seen before age 2 years with a median age of 6 to 8 years



Patient usually gives history of preceding streptococcal pharyngitis or cellulitis 1 to 3 weeks prior to presentation.

Presentations may range from asymptomatic hematuria to gross hematuria and proteinuria, hypertension, edema, and oliguria.

## Diagnosis

History of associated illness is helpful but not essential.

Laboratory values supporting diagnosis include low serum C3, elevated antistreptolysin O titer, urine red and white blood cell casts, hematuria, and proteinuria. Patients may also present with an elevated blood urea nitrogen, reduced glomerular filtration rate, and hypoalbuminemia may be present.

## Management

Management is primarily supportive because this usually self-resolves.

Decreased renal function or hypertension would be an indication for admission. Initiate salt and fluid restriction and possibly diuretic if the patient is edematous. Calcium channel blockers or intravenous hydralazine should be considered given degree of hypertension if present. Hypertension often resolves within 1 to 2 weeks.

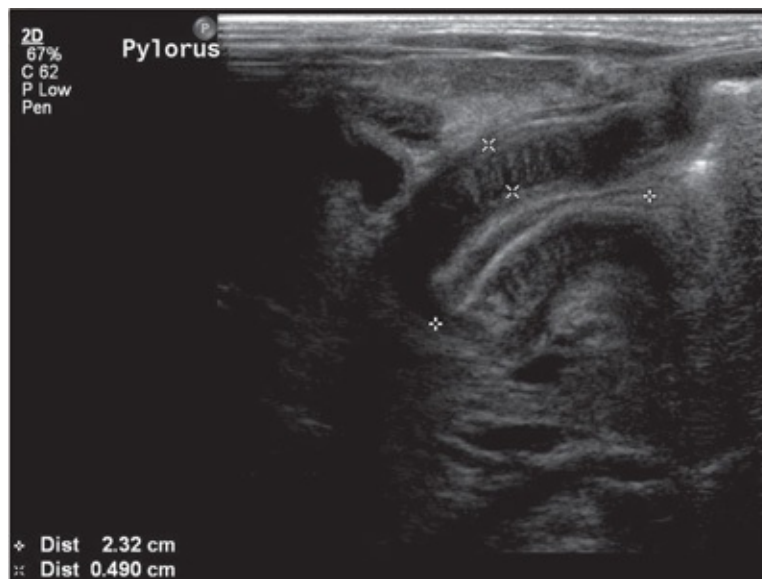
Clinical symptoms will often improve in 1 to 2 weeks; however, microscopic hematuria and proteinuria may persist for up to 6 months.

## 31. PYLORIC STENOSIS

Neil G. Uspal



**FIGURE 1.** Plain radiograph demonstrating distended stomach in a patient with pyloric stenosis.



**FIGURE 2.** Ultrasound demonstrating increased pylorus muscle thickness and length.

## Clinical Presentation

Progressive, increasingly forceful emesis, typically after feeds  
Most commonly presents in *3rd to 8th* week of life  
Nonbilious emesis—if patient has bilious (green) emesis, strongly consider alternative diagnosis.  
Classically eager to feed despite emesis  
Typically well-appearing but may be dehydrated or in hypovolemic shock if symptoms long standing

## Diagnosis

May be possible to palpate an olive-shaped mass in the epigastric region of the abdomen if the patient is relaxed  
Classic laboratory findings are a hypochloremic, hypokalemic metabolic alkalosis, although labs often normal. Patient may also be hyponatremic and/or have an elevated blood urea nitrogen and creatinine in more advanced cases.  
Pyloric ultrasound is the imaging modality of choice—accuracy of ultrasound is operator-dependent.  
On ultrasound, pylorus muscle *thickness*  $>3\text{ mm}$  and *length*  $15\text{ mm}$  is considered diagnostic.  
X-ray may demonstrate large, distended stomach. Upper GI also diagnostic, and shows additional anatomy but requires radiation—typical finding of thin, elongated “string” of contrast between the stomach and duodenum.

## Management

Initial management involves rehydration and correction of electrolyte abnormalities with isotonic saline (initial 20 mL/kg bolus).  
Maintenance fluids of D5.45 normal saline at 6 mL/kg/h should then begin, adding 20 mEq/L of potassium chloride to the fluids after observed urine output.  
Electrolyte abnormalities should be corrected before operative repair (persistent alkalosis may cause postoperative apnea and difficulty extubating).  
Consider placing a 10F Replogle nasogastric tube to reduce emesis and decompress the stomach prior to operative repair.

When electrolytes corrected, pyloric stenosis is corrected operatively by making a longitudinal incision in the pylorus to the submucosa. Surgery may be open or laparoscopic.

## 32. SALTER-HARRIS FRACTURE

Jaime K. Otilio, Ian Kane



**FIGURE 1.** Radiographs with Salter-Harris fractures.

### Clinical Presentation

In children, the physis (growth plate) is the last part of the bone to ossify, making it vulnerable to fracture.

Physeal fractures complicate up to 30% of pediatric long bone fractures. The Salter-Harris system is the most widely used classification system for physeal fractures.

Type I: fracture through the physis, separating the epiphysis from the metaphysis

Type II: fracture partially through the physis with extension into the metaphysis

Type III: fracture partially through the physis with extension into the epiphysis

Type IV: fracture through the epiphysis, physis, and metaphysis

Type V: crush injury to the physis resulting in later growth arrest

### Diagnosis

Most children present with localized pain and swelling after a traumatic event such as a fall.

Plain radiographs are generally diagnostic but may be negative with nondisplaced Salter-Harris fracture type I because the physis is radiolucent. Salter-Harris type V fractures are often not diagnosed until growth arrest becomes evident after an injury has occurred. Tenderness of the growth plate in a child is typically managed as a Salter-Harris type I fracture even if plain radiographs are negative.

## Management

Nondisplaced Salter-Harris type I and II fractures are typically treated with immobilization in a cast or splint.

Displaced Salter-Harris type I and II fractures require open or closed reduction and immobilization.

Salter-Harris type III and IV fractures usually require open reduction and internal fixation.

Salter-Harris type V fractures may need surgical intervention in addition to cast immobilization. There is almost always a growth arrest with these types of fractures.

Close orthopedic follow-up is vital for all physeal fractures due to the possibility of growth arrest or disruption at the fracture site.

### **33. SCARLET FEVER**

Robin Gehris, Jonathan Lewis



**FIGURE 1.** Strawberry tongue of scarlet fever.



**FIGURE 2.** Desquamation of the skin post scarlet fever.

#### **Clinical Presentation**

Scarlet fever is a nonsuppurative complication of group A streptococcal (GAS) infection.



Occurs in 10% of streptococcal pharyngitis cases  
Less commonly associated with GAS skin or soft tissue infection

## Diagnosis

History commonly includes fever, sore throat, headache, abdominal pain, and rash.

Diffuse, erythematous, sandpaper eruption (erythema may be lacking in dark-skinned individuals)

Secondary features may include erythematous skin folds in joints, strawberry tongue, and circumoral pallor.

Desquamation of skin may occur, typically as late finding, approximately 1 week after onset of diffuse rash.

GAS rapid assay or throat culture may be obtained but is unnecessary because diagnosis is clinical.

## Management

Rash begins to fade after 3 to 5 days without treatment; however, resolution is accelerated with antibiotics.

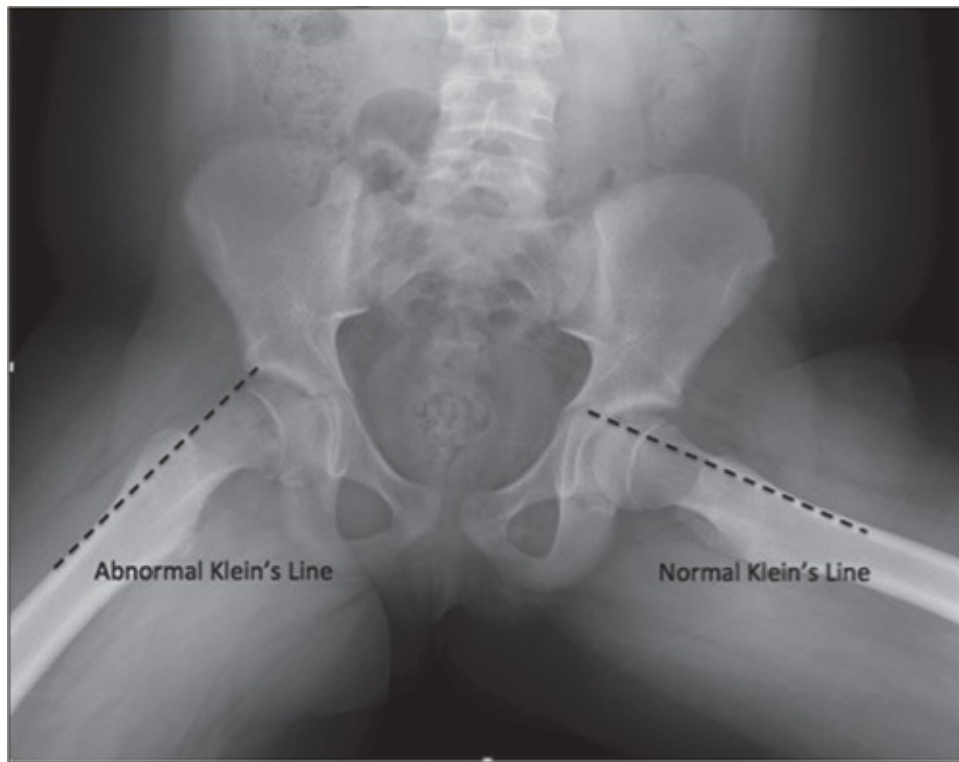
Treatment of scarlet fever is the same as that of streptococcal pharyngitis.

First-line therapy is penicillin or amoxicillin for 10 days.

Cephalosporin, macrolide, or clindamycin may be used in penicillin-allergic patients.

## 34. SLIPPED CAPITAL FEMORAL EPIPHYSIS

Maya Subbarao Iyer, Brian Schultz



**FIGURE 1.** Klein's lines are drawn over the superior portion of the femoral neck and should transect a portion of the epiphysis normally.

### Clinical Presentation

Presents with pain and altered gait

Most common demographic is the obese adolescent, and it is 3 times more common in males.

Patients will often complain of nonradiating, dull, aching pain in the hip, groin, thigh, or knee.

There is usually no preceding traumatic insult; however, the patient may recall minor trauma.

Cause of slipped capital femoral epiphysis (SCFE) is unknown.

## Diagnosis

Diagnosis is made on history as well as plain radiographs of the hips, including the anteroposterior (AP) and either cross-table lateral or frog-leg views.

AP radiographs may show blurring between the metaphysis and growth plate. The cross-table lateral view demonstrates the posterior displacement and step-off of the epiphysis on the femoral neck. This is recognized as the “falling ice cream off of the cone.”

Klein’s line passes outside of the epiphysis.

## Management

SCFE is an orthopedic emergency, and patients should be placed on bed rest and made non–weight-bearing.

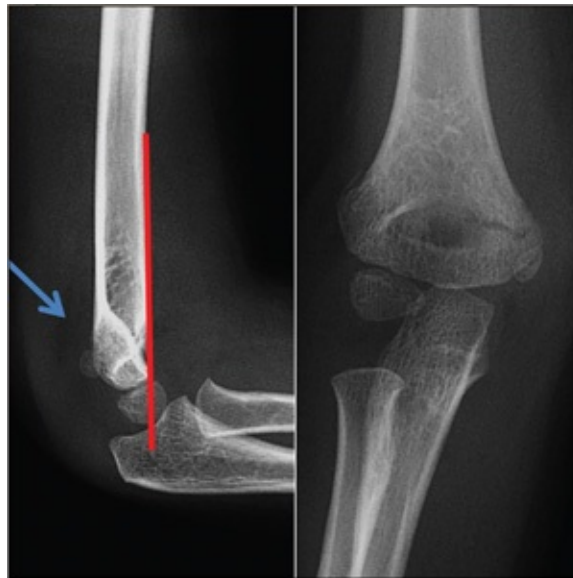
Management is surgical with pinning through the center of the epiphysis.

Complications include avascular necrosis and osteoarthritis.

Approximately 30% to 60% of patients will develop SCFE in the contralateral hip within 18 months, yet contralateral pinning remains controversial.

## 35. SUPRACONDYLAR FRACTURE

Desiree Noel Wagner Neville, Noel Zuckerbraun



**FIGURE 1.** Normal anterior humeral line (*red line*), but distended posterior fat pad (*blue arrow*) suggests fracture.



**FIGURE 2.** Abnormal anterior humeral line (*red line*) in a patient with a supracondylar fracture.

### Clinical Presentation

Supracondylar fractures account for the majority of elbow fractures in children.

Most common age is 3 to 10 years.

Most are caused by fall on an outstretched arm with hyperextension of the elbow.

Fall onto a flexed elbow is a much less common injury mechanism.

Presentation can vary from isolated tenderness to obvious swelling and deformity.

## Diagnosis

Full neurologic and vascular exams and splinting of deformed arm as it lies True lateral (with flexion at 90°) and anteroposterior radiographs confirm diagnosis.

Degree of displacement determines management and is defined by the anterior humeral line.

Anterior humeral line is drawn along the anterior margin of the humerus on the lateral view and should intersect the ossification center of the capitellum in the middle or posterior third in normal alignment.

Nondisplaced fractures can have only anterior and/or posterior fat pad elevation on radiographs with a subtle or nonvisible fracture line. A posterior fat pad is never normal.

## Management

Nondisplaced or minimally displaced fractures can be immobilized in a long arm splint with elbow flexed typically to no more than 90° with early orthopedic referral.

Displaced fractures and those with neurovascular compromise require emergent orthopedic consult for reduction.

Neurologic deficits are common—radial, median, and ulnar nerves can be affected.

Vascular spasm/compression that resolves with emergent reduction is more common than transection.

Admission is needed for displaced fracture to identify increased swelling and

compartment syndrome, which presents with pain out of a proportion as a common first symptom.

## 36. TODDLER'S FRACTURE

Susan Yaeger, Suzanne Schmidt



**FIGURE 1.** Subtle toddler's fracture on AP view (arrow).





**FIGURE 2.** A second patient with a more apparent Toddler's fracture (arrow).

## Clinical Presentation

Found in young, ambulatory children, ages 9 months to 3 years

Injury is often unwitnessed and occurs after a minor twist to the leg or accidental fall while running.

Children present with a limp or refusal to bear weight on the affected leg.

## Diagnosis

Exam with no obvious swelling or deformity

Patient may have tenderness to palpation over the mid to distal tibia or pain with gentle rotational stress to the lower leg.

Radiographs of the tibia and fibula show a nondisplaced oblique or spiral fracture of the distal third of the tibia.

Fracture may be subtle or not evident on initial radiographs. Healing callus is seen on repeat radiographs at 10 to 14 days.

In the case of negative films but high suspicion, diagnosis can be made

clinically if other causes (e.g., osteomyelitis, septic arthritis) can be excluded.

## Management

The fracture is treated with immobilization in a long-leg cast for 3 to 4 weeks. Toddler's fractures are accidental and alone are not suggestive of abuse. A spiral fracture of the leg in a *nonambulatory* child is concerning for inflicted injury, and further investigation (including skeletal survey) is indicated.

## 37. TORUS FRACTURE

Maria C. Antonucci, Suzanne Schmidt

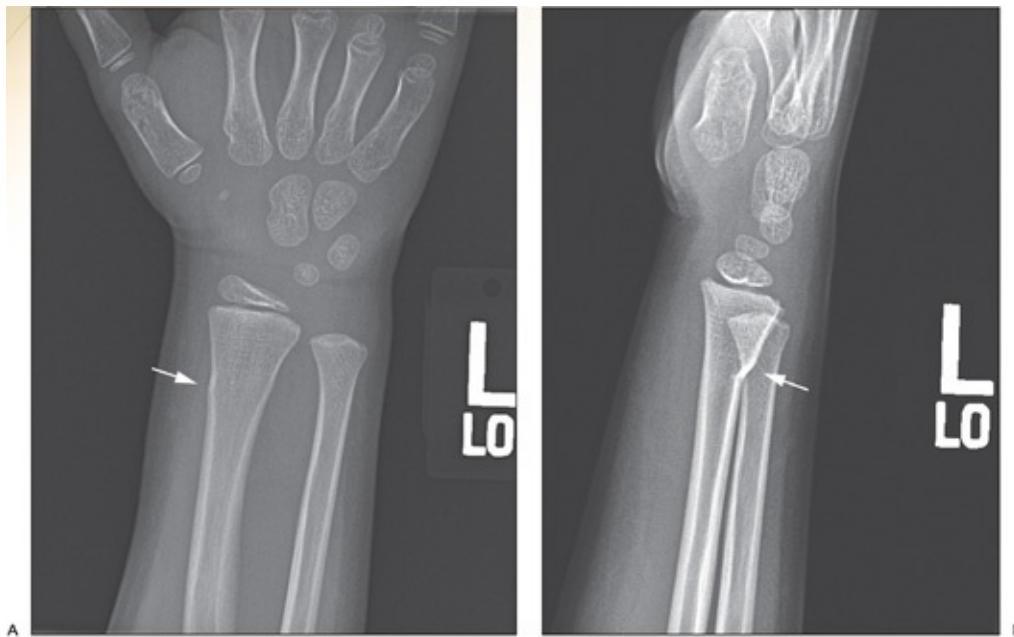


FIGURE 1. A: AP. B: Lateral radiographs of a torus fracture of the distal radius (arrows).

### Clinical Presentation

Common fracture in children due to porous metaphyses

An axial load to the extremity (fall on outstretched hand) causes a fracture at the junction of the metaphysis and diaphysis.

Most common location of injury is distal radius +/- ulna.

Patients may have delayed presentation because this fracture is typically less painful than other types of fractures.

### Diagnosis

Physical exam shows mild swelling at the fracture site, usually without gross deformity.

Look for point tenderness and pain with loading force or range of motion.

Two or three radiographic views of the affected area are indicated.

Subtle findings on anteroposterior view are often more apparent on lateral or oblique views.

## Management

Evaluate for neurovascular compromise, although rare in this fracture type.

Rule out growth plate involvement (Salter-Harris type II fracture).

Reduction is usually not indicated unless angulation is greater than  $15^{\circ}$ .

Stabilization for 3 to 4 weeks in prefabricated volar forearm splint is preferred over short arm cast due to comfort, convenience, and fewer complications.

## 38. TRANSIENT SYNOVITIS

Margaret B. Nguyen, Melissa A. Vitale



**FIGURE 1.** Transient synovitis of the left hip. Note the hip is flexed, abducted, and externally rotated.

### Clinical Presentation

Transient synovitis is the self-limited inflammation of the hip synovium commonly seen in children ages 3 to 10 years.

Affected children will present with the acute onset of hip or knee pain, antalgic gait, and preference to keep the hip flexed, abducted, and externally rotated.

Most children are afebrile.

There is often a history of preceding viral illness.

### Diagnosis

Transient synovitis is a clinical diagnosis as well as a diagnosis of exclusion; important differential diagnoses to consider are fracture, septic arthritis, and osteomyelitis.

Perform a log-roll test to quantify the degree of hip inflammation. With patient supine, distract the young child by pretending to examine the foot or knee.

Gently rotate the hip by grasping the foot or knee. Internal rotation of  $\geq 30^\circ$  without complaints of pain is reassuring for transient synovitis.

A trial with ibuprofen is recommended as patients with transient synovitis often return to weight bearing, which can support the diagnosis. Children with persistent hip pain or inability to bear weight after ibuprofen should be considered for further workup.

A child who appears ill, has high fever ( $>38.5^{\circ}\text{C}$ ), or markedly severe hip pain likely does not have transient synovitis, and alternative diagnoses should be considered.

Labs are not recommended or diagnostic for transient synovitis. However, in the case that alternative diagnoses are being considered such as septic arthritis, C-reactive protein  $<2$  mg/dL and erythrocyte sedimentation rate  $<20$  mm/h are supportive of a diagnosis of transient synovitis. Children with elevated inflammatory markers should undergo hip ultrasonography and be considered for diagnostic arthrocentesis.

## Management

The mainstay of management is supportive care with ibuprofen every 6 hours and activity as tolerated.

Resolution of symptoms generally occurs within 1 to 4 weeks.

Recurrence has been reported in up to 26% of patients with transient synovitis.

Symptoms persisting beyond 1 month should be evaluated for other hip pathology such as Legg-Calvé-Perthes disease.



SECTION

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TOXICOLOGIC

SECTION EDITOR  
Ernest Wang



# 1. CARBON MONOXIDE

Jerrold B. Leikin



**FIGURE 1.** Classic cherry red colored skin of carbon monoxide poisoning. (From McConnell TH. *The Nature of Disease Pathology for the Health Professions*. Philadelphia, PA: Lippincott Williams & Wilkins; 2007.)

## Clinical Presentation

Dull, frontal headache often associated with dizziness, emesis, weakness, retinal hemorrhage

Often associated with tachypnea and tachycardia

Frequently confused with viral syndrome or food poisoning

Multiple cohabitants' involvement and/or use of alternative heating sources are clues to diagnosis.

## Diagnosis

Carboxyhemoglobin levels are useful:

Normal nonsmoker: 0% to 5%

Normal smoker: 5% to 10%

Metabolic acidosis with elevated lactate can occur with severe exposure; respiratory alkalosis usually seen.

Basal ganglia lesions (bilateral) can be seen on magnetic resonance imaging.

## Management

100% oxygen provided by nonrebreather face mask or endotracheal tube

Cardiac monitoring

Hyperbaric oxygen (HBO) is treatment of choice in patients with severe carbon monoxide poisoning resulting in the following:

Syncope/coma

Seizures

Confusion/altered medical status

Prolonged carbon monoxide (CO) exposure (>24 hours)

Carboxyhemoglobin >25%

Age older than 35 years

Abnormal cerebellar function

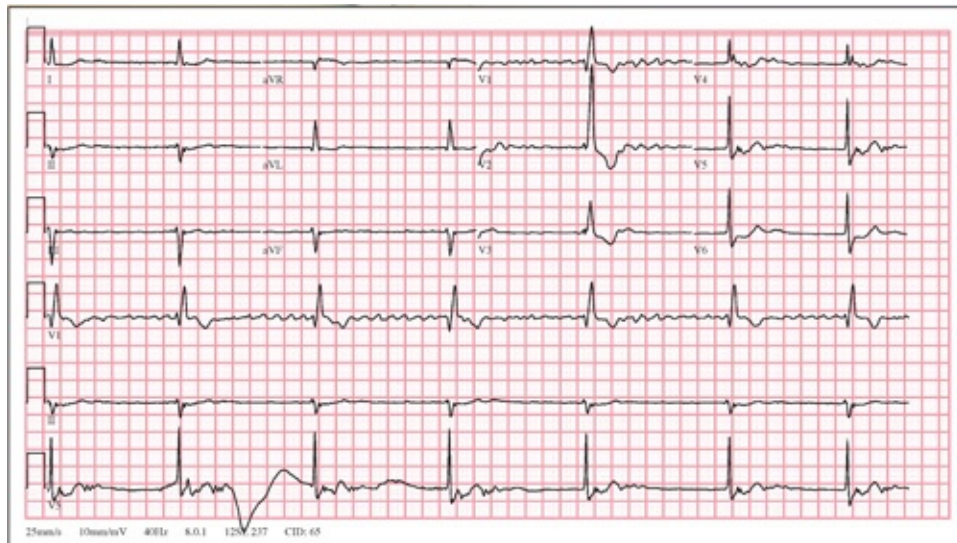
Fetal distress in pregnancy

Acute cardiac symptoms

HBO should be initiated within 6 hours.

## 2. CARDIAC GLYCOSIDE TOXICITY

Todd W. Thomsen



**FIGURE 1.** Digitalis toxicity resulting in a slow and regular atrial fibrillation with complete AV nodal blockage, leading to a junctional escape rhythm.

### Clinical Presentation

Cardiac glycosides, such as digitalis, have a narrow therapeutic index and a wide range of toxic effects.

Inhibition of the  $\text{Na}^+/\text{K}^+$  ATPase increases cardiac automaticity and may result in extrasystoles or tachycardia. Increased parasympathetic tone slows conduction in the sinoatrial and atrioventricular (AV) nodes and may result in bradycardia or heart block. Symptoms may include palpitations, syncope, or cardiovascular collapse.

Hyperkalemia is an ominous finding. Noncardiac toxicity includes gastrointestinal, visual (e.g., blurred vision, halos around points of light, and greenish-yellow visual discoloration), and neurologic symptoms.

### Diagnosis

Digitalis levels  $>2.0$  ng/mL are considered supratherapeutic. However, there is

poor correlation between serum digitalis level and clinical symptoms. Arrhythmias include bradycardia, complete heart block, premature ventricular complexes, atrial tachycardia with AV block, junctional tachycardia, and bidirectional ventricular tachycardia.

## Management

Gastrointestinal decontamination with activated charcoal or cholestyramine may be considered within the first 2 hours of ingestion. Multiple-dose activated charcoal may be beneficial (enterohepatic recirculation).

The antidote for severe digitalis toxicity is digoxin-specific antibody fragments (DSFab). Indications for DSFab administration include life-threatening cardiac arrhythmia (e.g., ventricular tachyarrhythmia), ingestion of massive quantities of digitalis (10 mg in adults, 4 mg in children), end-organ dysfunction (including altered mental status), and hyperkalemia (serum potassium >5 mEq/L).

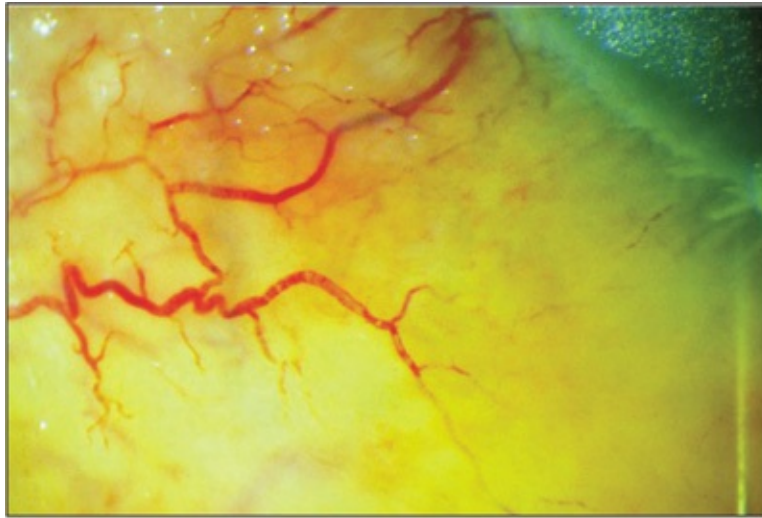
Hyperkalemia is best treated by the administration of DSFab. If unavailable, sodium bicarbonate and insulin and glucose may be used. Intravenous calcium was thought to exacerbate the poisoning, but recent reviews suggest it is most likely safe.

Supraventricular tachyarrhythmias may be treated with short-acting  $\beta$ -blockers (esmolol). Lidocaine for the treatment of ventricular tachyarrhythmias.

Atropine or pacing may be used for bradyarrhythmias. Contraindicated medications include calcium channel blockers and class IA antiarrhythmic agents such as procainamide.

### 3. CORNEAL BURNS—ALKALI

Jerrold B. Leikin



**FIGURE 1.** Chemical burn. High-magnification view of the eye demonstrates ischemia of the conjunctiva and sclera. There is segmentation of the red blood cells, indicating lack of blood flow. (From Rapuano CJ. *Wills Eye Institute—Cornea*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.)

#### Clinical Presentation

Most commonly in males ages 16 to 45 years old, usually occurring in industrial accidents or at home

Severity of burn is directly related to surface area of exposure.

Alkali compounds are more lipophilic and are able to penetrate more easily than acid substances due to cell membrane saponification. Hydrofluoric acid is the only acid that is known to penetrate readily.

#### Diagnosis

ROPER-HALL CLASSIFICATION			
Grade	Prognosis	Cornea	Conjunctival Limbus
I	Good	Corneal epithelial damage	No limbal ischemia
II	Good	Corneal haze and iris details visible	<1/3 limbal ischemia
III	Guarded	Total epithelial loss, stromal haze, and iris details obscured	1/3 to 1/2 limbal ischemia
IV	Poor	Corneal opaque and iris and pupil obscured	>1/2 limbal ischemia

## Management

Copious eye irrigation with normal saline drip through Morgan Lens system for at least 30 minutes

Check water runoff with pH litmus paper every 5 to 10 minutes until neutral pH is achieved.

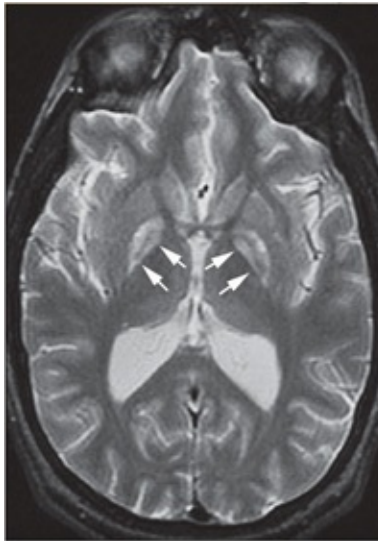
Subsequent care may include cycloplegics, topical antibiotics, pressure patch, oral pain medications, artificial tears, topical steroids, and intraocular pressure management.

Surgical debridement may be necessary for retained foreign objects.



## 4. CYANIDE POISONING

Jerrold B. Leikin



**FIGURE 1.** Axial T2-weighted image shows abnormal hyperintensity (white arrows) of the globi pallidi. (From Barkovich AJ, Raybaud C. *Pediatric Neuroimaging*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011.)



**FIGURE 2.** Urine of a patient being treated for smoke inhalation with hydroxocobalamin.



## Clinical Presentation

Cyanosis, dizziness, flushing, hypotension due to vasodilation, tachycardia followed by bradycardia, mydriasis, nystagmus

Seizures may occur followed by central nervous system depression.

Pulmonary edema may occur.

Bitter almond odor; fatal oral dose is 200 mg; fatal inhaled dose is 270 ppm.

## Diagnosis

Whole blood cyanide concentration (smoker  $\leq 0.5$  mg/L; flushing/tachycardia 0.5 to 1.0 mg/L; obtundation 1.0 to 2.5 mg/L; coma/death  $> 2.5$  mg/L)

When cyanide toxicity is suspected, a plasma lactate of  $> 8$  mm/L is 94% sensitive and 70% specific for blood cyanide concentration over 1 mg/L.

Anion gap metabolic acidosis

Elevated venous oxygen saturation (over 90%) due to impaired tissue extraction of oxygen (narrow arteriovenous oxygen difference)

## Management

May require decontamination if chemically exposed

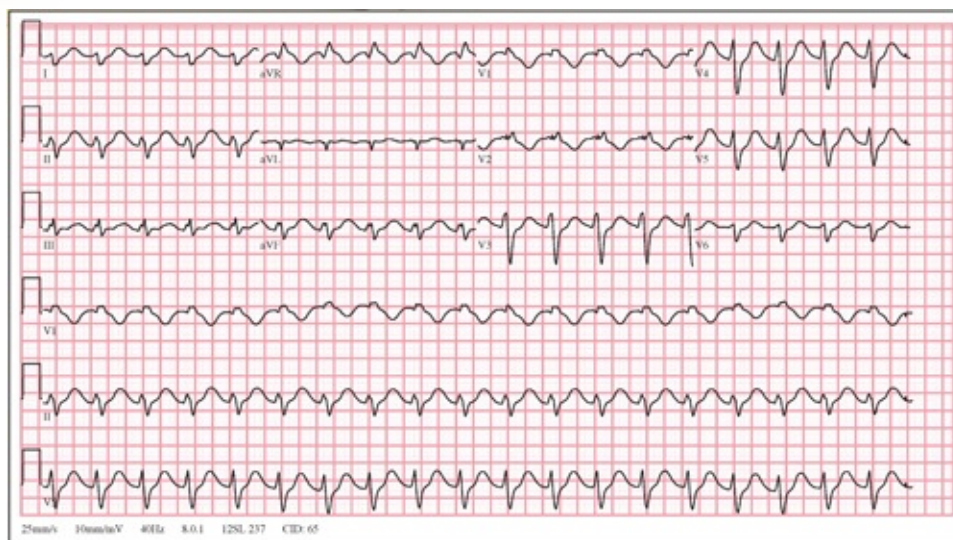
Oral ingestion: 1 g/kg of activated charcoal within 1 hour

Administer 100% oxygen.

Antidote: hydroxocobalamin. Dose is 70 mg/kg (usually 5 g) intravenous over 15 minutes; additional 5 g intravenous may be given (not to exceed cumulative dose of 10 g).

## 5. CYCLIC ANTIDEPRESSANTS

Todd W. Thomsen



**FIGURE 1.** ECG of a patient with severe CA poisoning. Note the presence of tachycardia, prolongation of all cardiac intervals, right axis deviation, and an increased R/S wave ratio in lead aVR.

### Clinical Presentation

Cyclic antidepressants (CAs) (e.g., amitriptyline) are extremely toxic in overdose and may lead to a myriad of clinical abnormalities, including death. The toxicity of the drug is mediated through both its anticholinergic and class Ia antiarrhythmic properties.

General symptoms are consistent with the anticholinergic toxidrome and include tachycardia, fever, mydriasis, dry skin and membranes, ileus, and altered mental status or delirium.

Cardiovascular effects include tachycardia, interval prolongation, right axis deviation, arrhythmia, and hypotension. Neurologic symptoms range from alterations in mental status to seizure and coma.

### Diagnosis

Clinical diagnosis is based on the history, physical exam, and

electrocardiogram (ECG).

Sinus tachycardia is the most common arrhythmia. Prolongation of all cardiac intervals (PR, QRS, and QT) may be encountered. QRS prolongation beyond 100 msec is associated with respiratory depression, seizures, and death. Right axis deviation ( $130^{\circ}$  to  $170^{\circ}$ ) is characteristic of CA toxicity. Careful examination of aVR is of prognostic value; an increased R/S wave ratio or a terminal R wave of  $\geq 3\text{mm}$  in this lead is associated with seizures and dysrhythmias.

## Management

Gastric decontamination with activated charcoal may be beneficial in patients presenting within 2 hours of ingestion. Enhanced elimination via dialysis is not beneficial due to the large volume of distribution of the CAs.

Intravenous sodium bicarbonate should be administered to patients with advanced symptoms (e.g., hypotension) or if the QRS is  $\geq 100$  msec.

The use of antiarrhythmic agents is generally discouraged because the cardiotoxic effects of the CAs are best treated with bicarbonate. If antiarrhythmics must be given, class Ib agents (e.g., lidocaine) are preferable. Pacing or chronotropic agents may be required for clinically substantial bradycardia.

Vasopressors may be required for refractory hypotension. Seizures are best treated with benzodiazepines.

There is growing evidence that intravenous lipid emulsion (ILE) may be effective in patients with severe CA toxicity. ILE is considered when symptoms are advanced; toxicology consultation is recommended.

## 6. INORGANIC LEAD POISONING

Jerrold B. Leikin



**FIGURE 1.** Lead poisoning with characteristic dense transverse metaphyseal lines (arrow). (From Chew FS. *Skeletal Radiology*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Microcytic anemia with basophilic stippling

Impaired renal function

Saturnine gout

Hypertension

Impaired new bone formation (increased metaphyseal density or “lead lines”)

Short stature

Impaired cognition/hearing at blood lead levels over 50  $\mu\text{g/dL}$

Constipation, anorexia, vomiting, colicky, abdominal pain, irritable behavior, lethargy at blood levels between 50 and 70  $\mu\text{g/dL}$

Encephalopathy, seizures, ataxia, apathy, cranial nerve palsies, persistent vomiting seen at blood lead levels over 70  $\mu\text{g/dL}$

## Diagnosis

Whole blood lead levels diagnostic for acute exposure

Erythrocyte protoporphyrin concentration usually elevated in severe toxicity

Renal function, urinalysis, and complete blood count, often abnormal

“Lead lines” at ends of long bones also may be caused by arsenic, bismuth, and mercury.

## Management

Exposure remediation

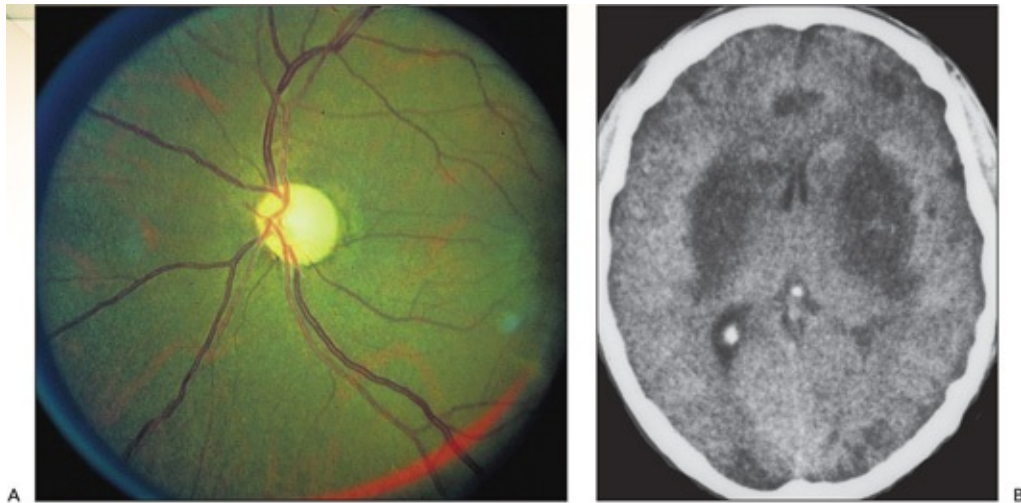
Chelation with succimer, calcium disodium ethylenediaminetetraacetic acid, or British anti-Lewisite

Seizure control with benzodiazepines

Gastrointestinal decontamination if radiopaque material noted on plain film (abdominal)

## 7. METHANOL POISONING

Jerrold B. Leikin



**FIGURE 1.** **A:** Bilateral optic disc pallor in a patient who lost vision after drinking methanol. **B:** Axial T2 after methanol ingestion shows swollen and bright basal ganglia and increased signal in most of the white matter. (**A**, Courtesy of Neil R. Miller, MD; **B**, From Castillo M. *Neuroradiology Companion: Methods, Guidelines, and Imaging Fundamentals*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

An industrial and household solvent; often found in windshield washer antifreeze fluid cleaners  
Metabolizes into formaldehyde and then is converted rapidly to formic acid, resulting in metabolic acidosis and ocular toxicity  
Blindness can occur after 10 mL ingestion; fatal dose is about 50 mL.  
Can be toxic by inhalation  
Reduction of serum pH and acidosis development may take 8 hours.  
Ocular effects include decreased visual acuity with edematous optic disc and photophobia.

### Diagnosis

Anion and osmolar gap acidosis  
Tachypnea present



Blood methanol level over 50 mg/dL associated with potential severe toxicity  
Peak serum concentration occurs within 1 hour.  
Bilateral putamen or globus pallidus abnormalities on brain imaging

## Management

Gastric lavage within 1 hour. Activated charcoal in a 5:1 ratio (charcoal:methanol) is of uncertain benefit.

Irrigate copiously for dermal or ocular exposure.

Treat acidosis with sodium bicarbonate (1 to 2 mEq/kg intravenous [IV]).

Leucovorin calcium or folic acid (1 mg/kg up to 50 mg every 4 hours) should be given to enhance formic acid metabolism.

Fomepizole to inhibit methanol metabolism: 15 mg/kg followed by 10 mg/kg IV every 12 hours for 4 doses

Hemodialysis if acidosis or renal failure develops, visual impairment is present, or if adult methanol consumption exceeds 40 mL; consider hemodialysis if blood methanol concentration exceeds 50 mg/dL.





SECTION

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ENVIRONMENTAL

SECTION EDITOR

Ernest Wang

# 1. BLACK WIDOW SPIDER BITE

Jerrold B. Leikin



**FIGURE 1.** A black widow spider. (Courtesy of Centers for Disease Control and Prevention in Schalock PC, Hsu JT, Arndt KA. *Lippincott's Primary Care Dermatology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

## Clinical Presentation

Spider exhibits a shiny black color with a red hourglass-shaped marking appearing on the abdomen of the female.

Found in all states except Alaska—usually in warmer climates

Most often located in corners of buildings, gardens, and trash piles

Female spider is 4 times larger than the male and is responsible for all toxic envenomations (alpha-latrotoxin).

Bites usually occur on the extremities.

## Diagnosis

Sharp local pain at bite site with an associated wheal and flare reaction

Muscle cramps usually begin within 1 hour, especially in the large muscle groups.

Abdominal rigidity and spasm without specific tenderness in about 50% of

patients

Fever is 10% to 15%. Headache in 5% to 25%. Vomiting and tachycardia can occur.

No confirmatory serologic test—laboratory data not useful, although leukocytosis often occurs.

## Management

Typical wound care

Calcium gluconate (10% mL per dose) may aid in treating muscle spasm, although benefit is not proven.

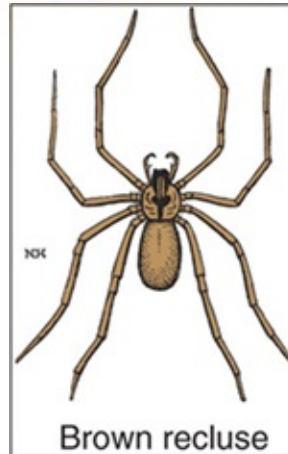
Intravenous opioids and benzodiazepines may be useful.

Antivenin may be available—usual therapeutic dose is one to two vials (intravenous) administered as 2.5 mL (one vial) in 50 to 100 mL 5% dextrose in water or normal saline over 20 to 30 minutes.

Mortality <1%

## 2. BROWN RECLUSE SPIDER BITE

Jerrold B. Leikin, Steven Aks



**FIGURE 1.** Brown recluse spider. (From Miller M, David Berry D. *Emergency Response Management for Athletic Trainers*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)



**FIGURE 2.** Brown recluse spider bite with thrombosis and necrosis. (From Craft N, Taylor E, Tumei PC, et al. *VisualDx: Essential Adult Dermatology*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Bites usually occur at night.

Dorsal cephalothorax has characteristic fiddle-shaped markings.

Range: South/Southwestern United States, Illinois, Iowa, Ohio, Nebraska, Missouri (Southern)

## Diagnosis

Mild erythema and pruritus can progress to severe skin ulceration.

Systemic findings (seen in 14% of bites) occur within 1 to 3 days and can include anemia, fever, dark urine, hemolysis, myopathy, vomiting, and thrombocytopenia.

The bite site can have a “halo” appearance with a small pustule surrounded by light pallor.

Wound can become necrotic in 3 days.

Death may be related to disseminated intravascular coagulation or renal failure.

No confirmatory blood test, although hemolysis, hematuria, leukocytosis, or coagulopathy may occur in severe envenomation.

## Management

Supportive therapy—elevate and immobilize affected limb.

Antihistamine may be given for pruritus.

Dapsone (100 mg twice daily for 2 weeks) may be useful if initiated within 8 hours of the bite.

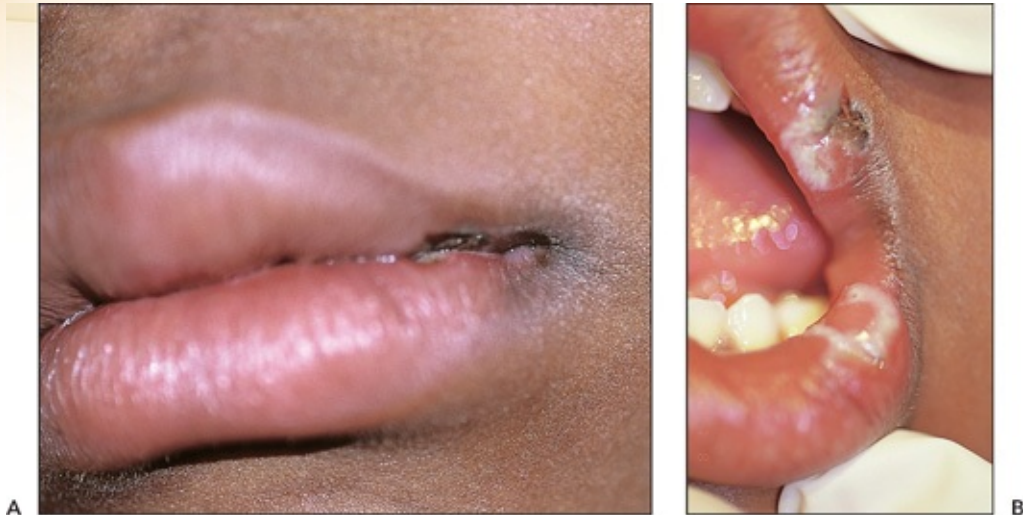
Dexamethasone, colchicine, or hyperbaric use is of unproven benefit.

Skin grafting may be required; negative pressure wound therapy using vacuum-assisted closure may improve healing.



### 3. ELECTRICAL INJURIES

Jerrold B. Leikin



**FIGURE 1.** A: This 5-year-old boy sustained a low voltage electrical burn when he bit an electrical cord. B: On follow-up after 5 days, only minimal improvement occurred, as is typical for electrical burns. (From Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)

#### Clinical Presentation

##### Low voltage

Alternating current is 3 times more dangerous than direct current.

Tissues with low resistance (nervous, muscle, mucous membranes, wet skin) are good conductors of electricity and thus more sensitive to adverse effects. Common when child chews on electrical cord where electricity arcs through saliva and results in burns to oral commissure and anterior tongue.

Oral burns susceptible to bleeding from superior labial artery in 10% of patients and can occur up to 2 weeks postinjury.

##### High voltage

Amperage of over 20 mA can cause respiratory arrest due to tetany of thoracic musculature; exposure to over 60 mA can cause ventricular fibrillation.

Partial-thickness dermal burns (especially noted on flexor surfaces of the hands); rhabdomyolysis is common.

Cataracts are seen in about 6% of high-voltage injuries.

## Diagnosis

Based on history and physical examination

Associated trauma with high voltage injuries

Complete blood count, complete metabolic profile, serum myoglobin/creatinine, phosphokinase with isoenzymes, electrocardiogram, and coagulation profile should be obtained.

## Management

Spinal immobilization may be indicated.

Control bleeding with manual pressure or, in severe cases, ligation of vessel.

Tetanus immunoprophylaxis

Cardiac monitoring for severe exposures

Cleanse the area; can treat externally with bacitracin

Admission to a burn unit



## 4. FROSTBITE

Jerrold B. Leikin



FIGURE 1. Frostbite of the fingers.

### Clinical Presentation

Peripheral cold injury affecting face, ears, hands, and feet

Blanching of skin and numbness usually noted on presentation. Blister formation can occur.

Pain is a dull ache—can become throbbing within 48 to 72 hours.

### Diagnosis

Favorable factors include retained sensation, normal skin color, and clear (rather than cloudy) blister fluid.

Poor prognostic factors include nonblanching cyanosis, firm skin, and dark fluid in blisters.

Tissue can mummify and demarcate within 60 to 90 days.

## Management

Warm water (40° to 42° C) immersion for 15 to 30 minutes; do not expose extremities to dry heat (i.e., heat lamp, heating pad). Do not rub or massage affected area.

Debride broken vesicles. Hemorrhagic blisters should not be debrided.

Apply topical aloe vera or antibiotic ointment. Use nonsteroidal antiinflammatory agents for pain.

Update tetanus status.

Avoid alcohol/tobacco products.

## 5. FOXGLOVE POISONING

Jerrold B. Leikin, Steven Aks



**FIGURE 1.** The foxglove plant (*Digitalis purpurea*). (From Fleisher GR, Ludwig S. *Textbook of Pediatric Emergency Medicine*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Due to ingestion of foxglove, digitalis purpurea. Ingestion of more than one leaf or one cup of tea may be toxic.

Gastrointestinal effects (vomiting) usually precede cardiovascular effects (bradycardia, prolonged PR interval, heart block) by several hours.

Hyperkalemia may occur.

Peak toxicity usually occurs within 12 hours.

### Diagnosis

Electrocardiogram may show heart block, bradycardia, prolonged PR interval, and decreased QT interval.

Digoxin and/or digitoxin assay will be positive although levels don't always correlate with degree of toxicity.

## Management

Activated charcoal (1 g/kg) or whole bowel irrigation is useful for gastric decontamination.

Bradycardia can respond to atropine.

Digoxin immune fab treatment is useful in treating hyperkalemia or severe bradycardia/heart block.

## 6. HIGH-PRESSURE INJECTION INJURIES

Clare Desmond



**FIGURE 1.** An innocuous-appearing puncture of the volar radial surface of the right small finger. This may be the only visible point of injury in a high-pressure injection injury (arrow). (From Wiesel SW. *Operative Techniques in Orthopaedic Surgery: Four Volume Set*. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.)

### Clinical Presentation

Patient will present with wound from a high-pressure injector usually in a digit.

Grease, paint, or hydraulic fluids are common agents of injury.

Oil-based paint causes the most severe reaction with higher risk of ischemia and amputation.

High-pressure injection can result in the deposition of foreign material significantly more proximal to the site of injury. An innocuous injury may result in significant damage.

### Diagnosis

Initially will appear benign with small area of injection with erythema and tenderness

Assess for neurologic and vascular compromise with frequent reexamination.

X-ray to assess for radiopaque substance, complicating fractures and subcutaneous air.

## Management

Pain control, but a digital block is contraindicated.

A common pitfall is underestimation of the extent of proximal injury.

Emergent orthopedic consult for surgical debridement and decompression is indicated. Long-term treatment may include surgical skin grafting.

Tetanus and antibiotic prophylaxis as indicated

## 7. HYDROFLUORIC ACID

Jerrold B. Leikin



FIGURE 1. Hydrofluoric acid burns on the dorsal (A) and volar hand (B).

### Clinical Presentation

Found in automotive cleaning products, glass etching, and used in the production of integrated circuits and computer screens

Route of exposure is usually dermal, but inhalation or ingestion can occur.

Latex gloves are not protective.

Severity of dermal injury is dependent on concentration of hydrofluoric acid solution.

Pain tends to be out of proportion compared to physical findings after dermal exposure.

Inhalation can cause bronchospasm, cough, laryngospasm, and airway edema (hemorrhagic).

Burns can develop; 3% total body surface area (TBSA) involvement may be fatal.

Fluoride anion combines with calcium and magnesium to cause profound hypocalcemia and hypomagnesemia; hyperkalemia may also develop.

### Diagnosis

Usually will present within hours of exposure

Electrocardiogram (ECG): prolonged QT interval with premature ventricular contractions secondary to hypocalcemia

Monitor ECG; electrolytes for hypercalcemia, hypomagnesemia, and



hyperkalemia.

Monitor for rhabdomyolysis.

## Management

Admit all dermal burn over 1% TBSA.

Immediate skin decontamination with copious amounts of water for at least 20 minutes following removal of all clothing, jewelry, shoes, etc.; pay close attention to skin folds, axillary/genital region, and feet.

Immediate eye decontamination with water, saline, or Ringer's Lactate using Morgan Lens for at least 20 minutes to conjunctival sac until pH = 7

Inhalation exposures should be given 100% oxygen.

Calcium gluconate topical application for dermal exposure

Intraarterial calcium gluconate (*not* calcium chloride) effective for thumb/finger/hand burns

Subcutaneous calcium gluconate (*not* calcium chloride) injection to affected dermal area may also be effective.

Treat hypocalcemia with intravenous or intraosseous calcium gluconate or calcium chloride.

## 8. HYPOTHERMIA

Jerrold B. Leikin, Ernest Wang



**FIGURE 1.** Affected extremities of hypothermic patient (89°F) receiving heated forced-air external rewarming.



**FIGURE 2.** Hypothermia. The Osborn waves are very prominent. (From Thaler MS. *The Only EKG Book You'll Ever Need*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.)

### Clinical Presentation

Classically defined as a reduction in the body's core temperature below 95.0°F (35.0°C)

Hypothermia may be classified as mild, moderate, or severe.

Risk factors for developing hypothermia include extremes of age (elderly people might not be able to remove themselves from cold environments, and young children lose heat more rapidly because of their increased total body surface area).

Accidental hypothermia is often related to environmental exposure, homelessness, concomitant trauma, psychiatric illness, and intoxicants.

## Diagnosis

A core body temperature should be obtained; usually rectal.

Evaluation includes complete blood count, electrolytes, electrocardiogram, and drug screen.

Multisystem involvement including hypotension, bradyarrhythmias, respiratory depression, altered mental status, shivering, peripheral vasoconstriction, cold diuresis

Evaluation for other causes including severe hypothyroidism (myxedema coma), acute spinal cord injury, diabetic ketoacidosis, multisystem trauma, and prolonged cardiac arrest and sepsis

## Management

*Mild hypothermia:* 32.2°C (90°F) to 35°C (95°F)—passive external rewarming; remove wet clothing. Cover patient with insulating material.

*Moderate hypothermia:* 28°C (82.4°F) to 32.2°C (90°F)—start active external rewarming; use radiant heat (thermal mattresses, electric heating blankets, or forced-air heating blankets). Administer warm humidified oxygen and intravenous saline.

*Severe hypothermia:* less than 28°C (82.4°F)—in addition to aggressive active core rewarming previously described, consider the following: bladder, colonic, and gastric irrigation; pleural cavity lavage; peritoneal lavage or dialysis; and extracorporeal warming or heated cardiopulmonary bypass. The complications of rewarming include arrhythmias, hypotension, core temperature after drop, and rhabdomyolysis.

Patients can be declared dead only after they are warm.

## 9. NAIL GUN INJURY

Jerrold B. Leikin



FIGURE 1. Nail gun injury through the gloved 3rd and 4th digits.



FIGURE 2. Radiograph demonstrating the nail.

### Clinical Presentation

Powered nail guns introduced in 1959

Powered by explosive charge or compressed air, the tool can fire nails up to 10 cm in length (equivalent to a .22-caliber handgun).

Sixteen percent to 22% of hand injuries occur at work.

## Diagnosis

Usually involves hands/feet

May be contaminated with oil, paper, or glue

Can involve injury to bone, tendon, nerves, or joint injury

Radiographs should be obtained with a minimum of two views.

## Management

Standard wound management with antibiotic treatment

Tetanus prophylaxis as indicated

Simple nail extraction can occur if there is no articular or neurovascular involvement.

Intraoperative exploration indicated with joint, tendon, and neurovascular involvement or if tissue is crushed.

## 10. POISON IVY

Jerrold B. Leikin



FIGURE 1. Poison ivy. (Image provided by Stedman's Medical Dictionary.)



FIGURE 2. Forearm of a patient with poison ivy.

### Clinical Presentation

From the plant *Toxicodendron radicans*. Contains an oily organic resin (urushiol) that can cause allergic contact dermatitis (type IV hypersensitivity).

Urticaria, pruritus, edema, and pain over affected area seen within 48 hours postexposure and peaks in 3 to 5 days  
Wash contaminated clothing, jewelry, tools, and even shoes/shoelaces.

## Diagnosis

No specific laboratory test

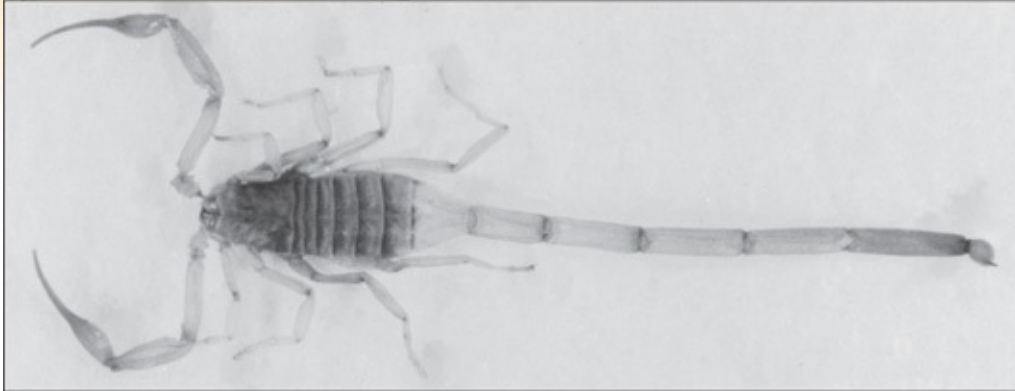
## Management

Wash affected area with soap and water within 15 minutes of exposure.  
Cool compresses or showers (soapless; warm but not hot water showers)  
Topical corticosteroid therapy cream, oral antihistamines, and/or prednisone (for large areas of exposure)  
Barrier creams (i.e., bentoquatam) absorb urushiol and can prevent or lessen dermal reactions.



## 11. SCORPION

Jerrold B. Leikin



**FIGURE 1.** Scorpion (*Centruroides exilicauda*). (From Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)

### Clinical Presentation

Scorpion (*Centruroides exilicauda*) has a body about 1 cm long with long thin pincers.

Found in Baja, California, and Southwestern United States

Usually found under rocks, logs, and tree bark; most active at night

Initially stings, producing intense local pain that peak at 5 hours

Systemic sequelae include sinus tachycardia, ataxia, abnormal ocular movements (33% to 58%), paresthesia, hypersalivation (21% to 35%), fasciculation, and ataxia often noted

Onset of symptoms within 1 hour

Mortality is rare in adults.

### Diagnosis

No specific blood test

### Management

Local wound care

Antiserum for severe envenomation is derived from goat serum and is available in Arizona for severe intravenous envenomations (cranial nerve or

somatic skeletal neuromuscular involvement); especially useful in children

Steroids not useful

Atropine can be used to dry up oral secretions. Intubation may be required.

Propranolol or phentolamine can be used to treat severely elevated blood pressure.

Continuous midazolam intravenous infusion can be considered in treating neuromuscular dysfunction (agitation, involuntary movements).

## 12. SNAKE BITES

Jerrold B. Leikin



**FIGURE 1.** Western coral snake. (From Fleisher GR, Ludwig S, Baskin MN. *Atlas of Pediatric Emergency Medicine*. Philadelphia, PA: Lippincott Williams & Wilkins; 2004.)

### Clinical Presentation

Coral snakes are brightly colored with red bands flanked by yellow bands that encircle the body, with a blunt and black head; found in Southwestern United States and Northern Mexico. Bite may be relatively painless. Symptoms may be delayed for hours and often include shortness of breath, ptosis, blurred vision, salivation, progressing to hypotension, numbness, and paralysis/seizures.

Rattlesnakes: found in all states except Maine, triangular head, vertically elliptical pupils, paired needle-like fangs. Venom causes local tissue damage, anticoagulant/platelet effects, and neurotoxic effects. Bite area becomes painful and edematous within minutes; edema and ecchymosis may spread throughout extremity within a few hours. Hemorrhagic blebs can appear within several hours. Rhabdomyolysis commonly encountered.

### Diagnosis

Coral snake: no specific blood test

Rattlesnake: Obtain coagulation parameters, complete metabolic profile, complete blood count, creatinine, phosphokinase, cardiac monitoring, urine

analysis, and electrocardiogram. Non-envenomation occurs in 10% to 50% of bites. Monitor for compartment syndrome.

## Management

Coral snake: requires 24-hour observation, 15% require intubation and ventilation, surgical debridement usually not necessary, no antivenin available

Rattlesnake: Observe 8 to 12 hours, maintain extremity in neutral position, tetanus prophylaxis, and local wound care. Venom extraction devices in prehospital care are not proven to be of benefit. Incision of fang and tourniquets marks is not recommended.

Antivenom with Crotalidae polyvalent immune fab should be considered for moderate (edema progression, hematologic abnormalities, or other systemic symptoms) or severe envenomations (marked progression of edema systemic signs, neurotoxic signs, hypotension, severe coagulopathy).

Blood products or fasciotomy rarely required

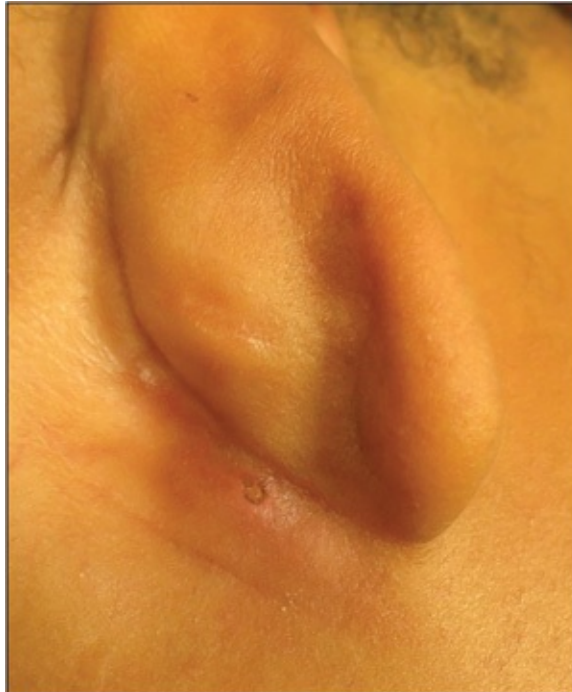
Poison center consultation; antivenom information hotline (1-877-377-3784) may be of assistance.

## 13. TICK BITES

Jerrold B. Leikin



**FIGURE 1.** Partially engorged tick.



**FIGURE 2.** Tick in Figure 1 was removed from behind the ear in a two-year-old 4 days after returning from a trip to Wisconsin.

## Clinical Presentation

Tick bites are responsible for transmitting Lyme disease, Rocky Mountain spotted fever, ehrlichiosis, babesiosis, anaplasmosis, tularemia, Colorado tick fever, and tick paralysis.

Tick species responsible include Ixodes, *Dermacentor*, and *Amblyomma*.

Ticks usually attach to the scalp. This is especially common in girls with long hair, where they go unnoticed, feed, and transmit disease.

## Diagnosis

Diagnosis is based on history of travel and symptoms if the tick is no longer present.

No specific blood or serum test is available in the emergency department.

## Management

Tick removal involves grasping the tick as close as possible to the skin surface with blunt curved forceps or tweezers while applying steady pressure without crushing the tick body.

Site should then be disinfected.

Removal of the entire tick is often curative in tick paralysis and supportive care is all that is needed.

Prophylaxis to prevent Lyme disease with a single dose of doxycycline 200 mg only if attached tick is identified as Ixodes (deer tick), tick has been attached for >36 hours, treatment begins within 72 hours of tick removal, rate of infection is >20 percent (New England, mid-Atlantic states, and Minnesota and Wisconsin), and doxycycline is not contraindicated (patient is not <8 years, pregnant, or lactating).



## 14. TRENCH FOOT

Morris Kharasch



**FIGURE 1.** Trench foot involving toes and entire soles of feet.

### Clinical Presentation

Caused by significant and prolonged exposure of the feet to a cold, moist, and wet environment

Can develop in as little as 13 hours in the right conditions

“Trench foot” or “immersion foot” was first ascribed to soldiers in World War I engaged in trench warfare while wearing wet, tight-fitting boots. Presently, it is a primary complaint in the undomesticated population.

One characteristic of trench foot that distinguishes it from frostbite is that it does not require freezing temperatures to occur (30° to 50°F).

### Diagnosis



Approximates the distribution of exposure; can involve toes, heel, or entire foot

The skin exhibits hyperemia in the early stages and subsequently can become pale and blotchy, and swelling.

In severe cases, hemorrhagic bullae, significant tissue loss, and even gangrene can occur.

Patients often complain of a loss of sensation, tingling, and numbness.

Pain, cramping, and distal motor weakness are also common.

## Management

Removal of any cold or moist clothing is the primary therapy on presentation.

Debride any dead tissues and open blisters in the emergency department.

Dry and elevate feet.

Apply clean dry socks and change daily.

Treat secondary cellulitis as indicated.

Long-term sequelae include prolonged and even lifelong sensitivity to cold weather in the affected distal extremity.

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